WEST Search History

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DATE: Wednesday, November 03, 2004

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L9	L8 AND N-kinase	0
☐	L8	435/7.1.CCLS.	8944
☐	L7	N-kinase dependent phosphorylation	2
☐	L6	(N-kinase)	3
☐	L5	Benowitz-L-I.IN.	9
☐	L4	Benowitz-Larry-I.IN.	15
☐	L3	Benowitz.IN.	51
☐	L2	Benowitz-L.IN.	0
☐	L1	(Benowitz-Larry.IN.)	0

END OF SEARCH HISTORY

h e b b cg b chh e f c e c ch

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Search Results - Record(s) 1 through 3 of 3 returned.

☐ 1. Document ID: US 20020160933 A1

Using default format because multiple data bases are involved.

L6: Entry 1 of 3 File: PGPB Oct 31, 2002

PGPUB-DOCUMENT-NUMBER: 20020160933
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020160933 A1

TITLE: Methods and compositions for producing a neurosalutary effect in a subject

PUBLICATION-DATE: October 31, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. Des

☐ 2. Document ID: US 20020160933 A1

L6: Entry 2 of 3 File: DWPI Oct 31, 2002

DERWENT-ACC-NO: 2003-328371
DERWENT-WEEK: 200331

TITLE: Producing neurosalutary effect, and treating neurological disorder, in a subject, by administering a therapeutically effective amount of a compound that modulates the activity of N-kinase, to the subject

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2001US-0949200 (September 7, 2001), 2000US-0656915 (September 7, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 20020160933 A1	October 31, 2002		020	A61K031/00

INT-CL (IPC): A61 K 31/00

ABSTRACTED-PUB-NO: US20020160933A
BASIC-ABSTRACT:

NOVELTY - Producing (M1) a neurosalutary effect in a subject, and treating a subject

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suffering from neurological disorder, involves administering a therapeutically effective amount of a compound (I) that modulates the activity of N-kinase, to the subject.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) identifying (M2) a compound capable of producing a neurosalutary effect in a subject, by contacting N-kinase or its biologically active fragment, with a test compound and determining the ability of the test compound to modulate the activity of N-kinase;

(2) a compound capable of producing a neurosalutary effect in a subject identified by the above method;

(3) an isolated N-kinase polypeptide (II) of the type that:

(a) is present in neonatal brain tissue

(b) is inhibited in the presence of 6-thioguanine

(c) is activated in the presence of Mn+2 but not by Mg+2 or Ca+2

(d) has a molecular weight of 49 kDa, and

(e) is eluted from a Cibacron Blue column at a NaCl concentration of 1.5-1.75 M;

(4) an antibody which is specifically reactive with an epitope of (II);

(5) a fragment of (II) comprising at least 15 contiguous amino acids, and capable of eliciting an immune response; and

(6) an isolated nucleic acid molecule (III) encoding a polypeptide comprising a sequence of 272 amino acids fully defined in the specification.

ACTIVITY - Anticonvulsant; Cerebroprotective; Neuroprotective; Nootropic.

No supporting biological data is given.

MECHANISM OF ACTION - Modulator of N-kinase activity (claimed); Promotes neuronal survival, axonal outgrowth and neuronal regeneration; Intracellular mediator of axonal outgrowth.

No supporting biological data is given.

USE - M1 is useful for producing a neurosalutary effect, and thus for treating a subject e.g. mammal, preferably human, suffering from neurological disorder such as spinal cord injury (including monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia), epilepsy, stroke and Alzheimer's disease. The treatment method further involves making a first assessment of a nervous system function prior to administering (I) and making a second assessment of a nervous system function after administering (I) to the subject. The nervous system function is a sensory function, cholinergic innervation or vestibulomotor function (claimed).

(II) is useful as bait protein in a two- or three-hybrid assay, to identify other proteins, which bind to or interact with N-kinase.

☐ 3. Document ID: JP 2004523470 W, WO 200220056 A2, AU 200187118 A, EP 1315514 A2

DERWENT-ACC-NO: 2002-393816
DERWENT-WEEK: 200451

TITLE: Producing a neurosalutary effect in a subject e.g., one suffering from neurological disorder such as stroke, to treat the subject, by administering a compound that modulates activity of N-kinase

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2000US-0656915 (September 7, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2004523470 W	August 5, 2004		077	A61K045/00
WO 200220056 A2	March 14, 2002	E	042	A61K045/00
AU 200187118 A	March 22, 2002		000	A61K045/00
EP 1315514 A2	June 4, 2003	E	000	A61K038/18

INT-CL (IPC): A61 K 9/10; A61 K 9/127; A61 K 38/18; A61 K 45/00; A61 P 9/10; A61 P 9/12; A61 P 25/00; A61 P 25/02; A61 P 25/08; A61 P 25/14; A61 P 25/16; A61 P 25/18; A61 P 25/24; A61 P 25/28; A61 P 43/00; C07 K 14/475; C07 K 16/40; C12 N 9/12; C12 N 15/09; C12 Q 1/48; G01 N 33/15; G01 N 33/50; G01 N 33/53; G01 N 33/566

ABSTRACTED-PUB-NO: WO 200220056A
BASIC-ABSTRACT:

NOVELTY - Producing (M1) a neurosalutary effect in a subject e.g., a subject suffering from a neurological disorder, to treat the subject suffering from the neurological disorder, involving administering to the subject a compound (I) that modulates the activity of N-kinase, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated N-kinase polypeptide (II) of the type that: is present in neonatal brain tissue; is inhibited in the presence of 6-thioguanine; is activated in the presence of Mn2+, but not by Mg2+ or Ca2+; has a molecular weight of approximately 49 kDa; and is eluted from a Cibacron Blue column at a sodium chloride concentration of 1.5-1.75 M;

(2) an antibody (III) which is specifically reactive with an epitope of (II);

(3) a fragment (IV) of (I), which comprises at least 15 contiguous amino acids, and is able to elicit an immune response;

(4) an isolated nucleic acid molecule that encodes (II); and

(5) a compound capable of producing a neurosalutary effect in a subject identified using (II).

ACTIVITY - Nootropic; neuroprotective; cerebroprotective; anticonvulsant; vulnerary; tranquilizer; antiparkinsonian; antimanic; antidepressant.

MECHANISM OF ACTION - N-kinase activity modulator; neuronal survival modulator; neuronal regeneration modulator; neuronal axonal outgrowth of central nervous system neurons e.g., retinal ganglion cells, modulator (all claimed).

No data given.

USE - (I) is useful for producing a neurosalutary effect in a subject e.g., a subject suffering from a neurological disorder, to treat the subject (preferably, humans)

f e h

suffering from the neurological disorder. The neurosalutary effect is produced by modulating neuronal survival, modulating neuronal regeneration or modulating neuronal axonal outgrowth of central nervous system neurons e.g., retinal ganglion cells, in a subject suffering from a neurological disorder such as spinal cord injury characterized by monoplegia, diplegia, paraplegia, hemoplegia and quadriplegia, or suffering from epilepsy, stroke or Alzheimer's disease.

(II) is useful for identifying a compound capable of producing a neurosalutary effect in a subject, preferably a compound which inhibits or stimulates the activity of N-kinase, which involves contacting (II) or its biologically active fragment with a test compound and determining the ability of the test compound to modulate the activity of N-kinase, thereby identifying a compound capable of producing a neurosalutary effect in a subject. The ability of the test compound to modulate the activity of N-kinase is determined by assessing the ability of the test compound to modulate N-kinase-dependant phosphorylation of a substrate. Optionally, (I) is identified using (II) by the following method which involves contacting (II) or its biologically active fragment, with a test compound, an N-kinase substrate (e.g., histone HF-1 protein), radioactive ATP (preferably gamma -32P), and Mn2+; and determining the ability of the test compound to modulate N-kinase dependent phosphorylation of the substrate, thereby identifying a compound capable of producing a neurosalutary effect in a subject. (II) used in the methods described above is preferably a recombinantly produced human N-kinase. Optionally, (II) is bovine N-kinase purified from a bovine source. The methods further involve determining the ability of the test compound to modulate axonal outgrowth of central nervous system neuron (all claimed).

(M1) is useful for treating a neurological disorder such as dementia's related to Alzheimer's disease, Parkinson's disease, senile dementia, Huntington's disease, Creutzfeldt-Jakob disease, Korsakoff's psychosis, mania, anxiety disorders, obsessive-compulsive disorder, anxiety, bipolar affective disorder. The methods are useful for preventing or treating neurological deficits in embryos or fetuses in utero, in premature infants, or in children with need of such treatment, including those with neurological birth defects. (I) is also useful for modulating activity of N-kinase, in vitro to modulate axonal outgrowth in vitro.

Terms	Documents
(N-kinase)	3

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Search Results - Record(s) 1 through 2 of 2 returned.

☐ 1. Document ID: US 20020160933 A1

Using default format because multiple data bases are involved.

L7: Entry 1 of 2 File: PGPB Oct 31, 2002

PGPUB-DOCUMENT-NUMBER: 20020160933
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020160933 A1

TITLE: Methods and compositions for producing a neurosalutary effect in a subject

PUBLICATION-DATE: October 31, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Des

☐ 2. Document ID: JP 2004523470 W, WO 200220056 A2, AU 200187118 A, EP 1315514 A2

L7: Entry 2 of 2 File: DWPI Aug 5, 2004

DERWENT-ACC-NO: 2002-393816
DERWENT-WEEK: 200451

TITLE: Producing a neurosalutary effect in a subject e.g., one suffering from neurological disorder such as stroke, to treat the subject, by administering a compound that modulates activity of N-kinase

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2000US-0656915 (September 7, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2004523470 W	August 5, 2004		077	A61K045/00
WO 200220056 A2	March 14, 2002	E	042	A61K045/00
AU 200187118 A	March 22, 2002		000	A61K045/00
EP 1315514 A2	June 4, 2003	E	000	A61K038/18

INT-CL (IPC): A61 K 9/10; A61 K 9/127; A61 K 38/18; A61 K 45/00; A61 P 9/10; A61 P

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9/12; A61 P 25/00; A61 P 25/02; A61 P 25/08; A61 P 25/14; A61 P 25/16; A61 P 25/18; A61 P 25/24; A61 P 25/28; A61 P 43/00; C07 K 14/475; C07 K 16/40; C12 N 9/12; C12 N 15/09; C12 Q 1/48; G01 N 33/15; G01 N 33/50; G01 N 33/53; G01 N 33/566

ABSTRACTED-PUB-NO: WO 200220056A
BASIC-ABSTRACT:

NOVELTY - Producing (M1) a neurosalutary effect in a subject e.g., a subject suffering from a neurological disorder, to treat the subject suffering from the neurological disorder, involving administering to the subject a compound (I) that modulates the activity of N-kinase, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated N-kinase polypeptide (II) of the type that: is present in neonatal brain tissue; is inhibited in the presence of 6-thioguanine; is activated in the presence of Mn2+, but not by Mg2+ or Ca2+; has a molecular weight of approximately 49 kDa; and is eluted from a Cibacron Blue column at a sodium chloride concentration of 1.5-1.75 M;

(2) an antibody (III) which is specifically reactive with an epitope of (II);

(3) a fragment (IV) of (I), which comprises at least 15 contiguous amino acids, and is able to elicit an immune response;

(4) an isolated nucleic acid molecule that encodes (II); and

(5) a compound capable of producing a neurosalutary effect in a subject identified using (II).

ACTIVITY - Nootropic; neuroprotective; cerebroprotective; anticonvulsant; vulnerary; tranquilizer; antiparkinsonian; antimanic; antidepressant.

MECHANISM OF ACTION - N-kinase activity modulator; neuronal survival modulator; neuronal regeneration modulator; neuronal axonal outgrowth of central nervous system neurons e.g., retinal ganglion cells, modulator (all claimed).

No data given.

USE - (I) is useful for producing a neurosalutary effect in a subject e.g., a subject suffering from a neurological disorder, to treat the subject (preferably, humans) suffering from the neurological disorder. The neurosalutary effect is produced by modulating neuronal survival, modulating neuronal regeneration or modulating neuronal axonal outgrowth of central nervous system neurons e.g., retinal ganglion cells, in a subject suffering from a neurological disorder such as spinal cord injury characterized by monoplegia, diplegia, paraplegia, hemoplegia and quadriplegia, or suffering from epilepsy, stroke or Alzheimer's disease.

(II) is useful for identifying a compound capable of producing a neurosalutary effect in a subject, preferably a compound which inhibits or stimulates the activity of N-kinase, which involves contacting (II) or its biologically active fragment with a test compound and determining the ability of the test compound to modulate the activity of N-kinase, thereby identifying a compound capable of producing a neurosalutary effect in a subject. The ability of the test compound to modulate the activity of N-kinase is determined by assessing the ability of the test compound to modulate N-kinase-dependant phosphorylation of a substrate. Optionally, (I) is identified using (II) by the following method which involves contacting (II) or its biologically active fragment, with a test compound, an N-kinase substrate (e.g., histone HF-1 protein), radioactive ATP (preferably gamma -32P), and Mn2+; and determining the ability of the test compound to modulate N-kinase dependent phosphorylation of the substrate, thereby identifying a compound capable of producing a neurosalutary effect in a subject. (II) used in the methods described above is preferably a recombinantly produced human N-kinase. Optionally, (II) is bovine N-kinase purified from a bovine source. The methods further involve determining the

ability of the test compound to modulate axonal outgrowth of central nervous system neuron (all claimed).

(M1) is useful for treating a neurological disorder such as dementia's related to Alzheimer's disease, Parkinson's disease, senile dementia, Huntington's disease, Creutzfeldt-Jakob disease, Korsakoff's psychosis, mania, anxiety disorders, obsessive-compulsive disorder, anxiety, bipolar affective disorder. The methods are useful for preventing or treating neurological deficits in embryos or fetuses in utero, in premature infants, or in children with need of such treatment, including those with neurological birth defects. (I) is also useful for modulating activity of N-kinase, in vitro to modulate axonal outgrowth in vitro.

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Terms	Documents
N-kinase dependent phosphorylation	2

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Search Results - Record(s) 1 through 51 of 51 returned.

☐ 1. Document ID: US 20040014710 A1

Using default format because multiple data bases are involved.

L3: Entry 1 of 51 File: PGPB Jan 22, 2004

PGPUB-DOCUMENT-NUMBER: 20040014710
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040014710 A1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

PUBLICATION-DATE: January 22, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/45; 514/263.37

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc

☐ 2. Document ID: US 20030236847 A1

L3: Entry 2 of 51 File: PGPB Dec 25, 2003

PGPUB-DOCUMENT-NUMBER: 20030236847
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030236847 A1

TITLE: Technology enhanced communication authorization system

PUBLICATION-DATE: December 25, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Joseph C.	Salt Lake City	UT	US	
Bunch, Kyle J.	Salt Lake City	UT	US	

US-CL-CURRENT: 709/206; 713/202

ABSTRACT:

A method of authorizing communications includes receiving a communication from a sender, determining if the communication contains a valid authorization code, notifying the sender if a valid authorization code is not detected with instructions on obtaining a valid authorization code and providing the sender with a service for

obtaining a valid authorization code in order to resend the communication with the valid authorization code. The method of authorizing communications also includes forwarding the communication to a recipient if a valid authorization code is detected and holding the communication in an unauthorized box if a valid authorization code is not detected.

☐ 3. Document ID: US 20030153501 A1

L3: Entry 3 of 51 File: PGPB Aug 14, 2003

PGPUB-DOCUMENT-NUMBER: 20030153501
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030153501 A1

TITLE: Methods and compositions for treating ocular disorders

PUBLICATION-DATE: August 14, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton	MA	US	

US-CL-CURRENT: 514/12

ABSTRACT:

The present invention provides a method for treating and/or preventing damage to a retina or optic nerve in a subject comprising administering to the subject a therapeutically effective amount of oncomodulin. Preferably, the subject is a mammal, most preferably, a human. In preferred embodiments, the oncomodulin may be used in combination with mannose, a mannose derivative and/or inosine.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc

☐ 4. Document ID: US 20020160933 A1

L3: Entry 4 of 51 File: PGPB Oct 31, 2002

PGPUB-DOCUMENT-NUMBER: 20020160933
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020160933 A1

TITLE: Methods and compositions for producing a neurosalutary effect in a subject

PUBLICATION-DATE: October 31, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/1

ABSTRACT:

Methods and compositions for producing a neurosalutary effect in a subject are provided. These methods generally involve administering to a subject a therapeutically effective amount of a compound that modulates the activity of N-kinase, or analog thereof. Pharmaceutical and packaged formulations including the compounds of the invention, e.g., compounds that modulate the activity of N-kinase, are also provided.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. Des

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☐ 5. Document ID: US 20020137721 A1

L3: Entry 5 of 51 File: PGPB Sep 26, 2002

PGPUB-DOCUMENT-NUMBER: 20020137721
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020137721 A1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

PUBLICATION-DATE: September 26, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/45

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. Des

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☐ 6. Document ID: US 20020128223 A1

L3: Entry 6 of 51 File: PGPB Sep 12, 2002

PGPUB-DOCUMENT-NUMBER: 20020128223
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020128223 A1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

PUBLICATION-DATE: September 12, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/45

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

☐ 7. Document ID: US 20020119923 A1

L3: Entry 7 of 51 File: PGPB Aug 29, 2002

PGPUB-DOCUMENT-NUMBER: 20020119923
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020119923 A1

TITLE: Methods and compositions for producing a neurosalutary effect in a subject

PUBLICATION-DATE: August 29, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Square	MA	US	

US-CL-CURRENT: 514/12; 514/47, 514/729

ABSTRACT:

Methods and compositions for producing a neurosalutary effect in a subject, such as modulating neuronal survival and/or regeneration in a subject, are provided. Pharmaceutical and packaged formulations are also provided.

☐ 8. Document ID: US 20020055484 A1

L3: Entry 8 of 51 File: PGPB May 9, 2002

PGPUB-DOCUMENT-NUMBER: 20020055484
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020055484 A1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

PUBLICATION-DATE: May 9, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/45

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

☐ 9. Document ID: US 20020042390 A1

L3: Entry 9 of 51 File: PGPB Apr 11, 2002

PGPUB-DOCUMENT-NUMBER: 20020042390
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020042390 A1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

PUBLICATION-DATE: April 11, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/45; 514/263.37

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

☐ 10. Document ID: US 6551612 B2

L3: Entry 10 of 51 File: USPT Apr 22, 2003

US-PAT-NO: 6551612
DOCUMENT-IDENTIFIER: US 6551612 B2

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

DATE-ISSUED: April 22, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Benowitz; Larry I.	Newton Centre	MA		

US-CL-CURRENT: 424/450; 424/422, 424/423, 424/484, 424/486, 424/489, 424/490, 424/497, 514/45

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

11 Claims, 16 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 9

☐ 11. Document ID: US 6440455 B1

L3: Entry 11 of 51 File: USPT Aug 27, 2002

US-PAT-NO: 6440455
DOCUMENT-IDENTIFIER: US 6440455 B1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

DATE-ISSUED: August 27, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Benowitz; Larry I.	Newton Centre	MA		

US-CL-CURRENT: 424/450; 424/422, 424/423, 424/484, 424/486, 424/489, 424/490,

424/497, 514/45

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

11 Claims, 16 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 9

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Des

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☐ 12. Document ID: US 6034247 A

L3: Entry 12 of 51 File: USPT Mar 7, 2000

US-PAT-NO: 6034247
DOCUMENT-IDENTIFIER: US 6034247 A

TITLE: Oxazolidinones and methods for the synthesis and use of same

DATE-ISSUED: March 7, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Smith, III; Amos B.	Merion	PA		
Hirschmann; Ralph F.	Blue Bell	PA		
Sprengeler; Paul A.	Philadelphia	PA		
Benowitz; Andrew B.	Willow Grove	PA		
Favor; David A.	Glenside	PA		

US-CL-CURRENT: 548/228

ABSTRACT:

Synthetic methods for pyrrolinone-based compounds are provided. Such compounds mimic or inhibit the biological and/or chemical activity of peptides.

5 Claims, 14 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 14

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Des

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☐ 13. Document ID: US 5898066 A

L3: Entry 13 of 51 File: USPT Apr 27, 1999

US-PAT-NO: 5898066
DOCUMENT-IDENTIFIER: US 5898066 A

TITLE: Trophic factors for central nervous system regeneration

DATE-ISSUED: April 27, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Benowitz; Larry I.	Newton	MA		
Irwin; Carleen A.	Newton	MA		
Jackson; Paul	Brookline	MA		

US-CL-CURRENT: 530/300; 530/399

ABSTRACT:

Cell culture conditions were developed which maintain the nerve cells of the retina in well-defined, serum-free conditions. The molecular factors that stimulate axonal regeneration from these neurons were characterized. The glial sheath cells that surround the axons of the optic nerve release two molecules that trigger and sustain nerve regeneration. One of the molecules is referred to as axogenesis factor 1 (AF-1), and is a low molecular weight polypeptide with a size in the range of 1000 daltons. The second molecule, AF-2, is a larger protein with a size of approximately 12,000 daltons. Studies indicate that these factors are strongly involved in CNS regeneration, and are therefore useful in the treatment of spinal cord and other nervous tissue damage.

1 Claims, 12 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 5

☐ 14. Document ID: US 5534849 A

L3: Entry 14 of 51 File: USPT Jul 9, 1996

US-PAT-NO: 5534849
DOCUMENT-IDENTIFIER: US 5534849 A

TITLE: Time multiplexed, false alarm resistant magnetically actuated security system

DATE-ISSUED: July 9, 1996

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
McDonald; Kevin B.	Portland	OR		
Pierce; Stephen W.	Tigard	OR		
Benowitz; Michael	Lake Oswego	OR		
Terrett; David S.	Beaverton	OR		

US-CL-CURRENT: 340/517; 340/511, 340/547, 340/551

ABSTRACT:

A magnetically actuated switch system (10) has reduced sensitivity to false alarms and reduced power consumption and resistance to physical and magnetic tampering. The system includes a signal processing circuit controlled by a microprocessor (16) that selectively enables magnetic sensors (12, 38), and compares the corresponding sensor outputs (76) to automatically calibrated level and predetermined timing characteristics.

16 Claims, 8 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 8

☐ 15. Document ID: US 3892658 A

L3: Entry 15 of 51 File: USPT Jul 1, 1975

US-PAT-NO: 3892658
DOCUMENT-IDENTIFIER: US 3892658 A

TITLE: Magnetic pulley for removal of non-magnetic pieces from waste material

DATE-ISSUED: July 1, 1975

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Benowitz; Sander	Sunnyvale	CA		

US-CL-CURRENT: 209/213; 100/91, 209/219, 209/223.2, 241/DIG.38

ABSTRACT:

A technique of removing electrically conductive nonmagnetic particles from a stream of material by a cylindrical magnet driven by a conveyor belt carrying the stream of material wherein attractor windings are provided in conjunction with excitation windings for forming zones of attraction of electrically conductive non-magnetic particles to the magnet. Electrically non-conductive and non-magnetic particles are thus not attracted to the magnet and are free to follow a different path than are the electrically conductive non-magnetic particles. Separation is thus achieved between them.

12 Claims, 6 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 4

☐ 16. Document ID: US 3824516 A

L3: Entry 16 of 51 File: USPT Jul 16, 1974

US-PAT-NO: 3824516
DOCUMENT-IDENTIFIER: US 3824516 A
**** See image for Certificate of Correction ****

TITLE: ELECTROMAGNETIC MATERIAL HANDLING SYSTEM UTILIZING OFFSET POLE SPACING

DATE-ISSUED: July 16, 1974

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Benowitz; Sander	Sunnyvale	CA	94087	

US-CL-CURRENT: 335/284; 198/619, 209/212, 241/DIG.38, 335/289

ABSTRACT:

A technique of moving electrically conductive non-magnetic particles wherein a plurality of electromagnets are positioned on either side of an air gap with each electromagnet facing a non-magnetic space between electromagnets on the opposite side of the air gap. The electromagnets are energized with polyphase current in a manner to generate a sweeping magnetic flux down the air gap for moving particles therealong. Eddy currents generated by one magnetic field relative phase reacts with flux of another magnetic field relative phase to provide motion of the article. Two specific utilizations of this technique are described; the separation of conductive non-magnetic particles from waste material and the movement of aluminum can lids.

13 Claims, 9 Drawing figures Number of Drawing Sheets: 3

☐ 17. Document ID: US 3614317 A

L3: Entry 17 of 51 File: USPT Oct 19, 1971

US-PAT-NO: 3614317
DOCUMENT-IDENTIFIER: US 3614317 A

TITLE: THREE-STATE FREQUENCY SHIFT SIGNAL RECEIVER

DATE-ISSUED: October 19, 1971

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Benowitz; Paul	Freehold	NJ		
Kahlbrock; Heinz	Edison	NJ		

US-CL-CURRENT: 375/335; 375/317

ABSTRACT:

A center frequency region in the signal channel band is assigned to supervisory (on-hook) signals and an upper and a lower frequency region is assigned to (mark and space) data signals whereby a greater frequency swing for increased power and bandwidth is obtained for the data signals. When incoming signals are in the center frequency region, the data signal output is blocked. Normal data signals, however, sweep through the center frequency region when a signal transition occurs. The data

signal is delayed so that the delayed signal transition appears in the output after the blockage terminates.

13 Claims, 3 Drawing figures Number of Drawing Sheets: 3

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Des

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☐ 18. Document ID: US 3466397 A

L3: Entry 18 of 51 File: USPT Sep 9, 1969

US-PAT-NO: 3466397
DOCUMENT-IDENTIFIER: US 3466397 A

TITLE: CHARACTER AT A TIME DATA MULTIPLEXING SYSTEM [TEXT AVAILABLE IN USOCR DATABASE]

DATE-ISSUED: September 9, 1969

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
IGNATOWITZ MICHAEL				
TUTELMAN DAVID M				
BENOWITZ PAUL				

US-CL-CURRENT: 370/479; 370/246, 370/501, 370/503, 370/522

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Des

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☐ 19. Document ID: US 3443022 A

L3: Entry 19 of 51 File: USPT May 6, 1969

US-PAT-NO: 3443022
DOCUMENT-IDENTIFIER: US 3443022 A

TITLE: HUB COUPLING SYSTEM [TEXT AVAILABLE IN USOCR DATABASE]

DATE-ISSUED: May 6, 1969

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
BENOWITZ PAUL				
KAHLBROCK HEINZ				

US-CL-CURRENT: 178/2R; 327/1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Des

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☐ 20. Document ID: US 2877456 A

L3: Entry 20 of 51 File: USPT Mar 10, 1959

US-PAT-NO: 2877456
DOCUMENT-IDENTIFIER: US 2877456 A

TITLE: Zero speed detector [TEXT AVAILABLE IN USOCR DATABASE]

DATE-ISSUED: March 10, 1959

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
SANDER BENOWITZ				

US-CL-CURRENT: 361/22; 324/161, 324/772, 340/671

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc

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☐ 21. Document ID: US 2344963 A

L3: Entry 21 of 51 File: USPT Mar 28, 1944

US-PAT-NO: 2344963
DOCUMENT-IDENTIFIER: US 2344963 A

TITLE: Shoelace [TEXT AVAILABLE IN USOCR DATABASE]

DATE-ISSUED: March 28, 1944

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
AARON BENOWITZ				

US-CL-CURRENT: 24/713; 24/712

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc

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☐ 22. Document ID: US 2185425 A

L3: Entry 22 of 51 File: USPT Jan 2, 1940

US-PAT-NO: 2185425
DOCUMENT-IDENTIFIER: US 2185425 A

TITLE: One-piece adjustable strap [TEXT AVAILABLE IN USOCR DATABASE]

DATE-ISSUED: January 2, 1940

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
BENOWITZ MAX A				

US-CL-CURRENT: 2/237

☐ 23. Document ID: US 1934398 A

L3: Entry 23 of 51 File: USPT Nov 7, 1933

US-PAT-NO: 1934398
DOCUMENT-IDENTIFIER: US 1934398 A

TITLE: Manufacturing of yarn [TEXT AVAILABLE IN USOCR DATABASE]

DATE-ISSUED: November 7, 1933

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
AARON BENOWITZ				

US-CL-CURRENT: 28/144

☐ 24. Document ID: US 1872389 A

L3: Entry 24 of 51 File: USPT Aug 16, 1932

US-PAT-NO: 1872389
DOCUMENT-IDENTIFIER: US 1872389 A

TITLE: Process for manufacturing a trimming material or cloth [TEXT AVAILABLE IN USOCR DATABASE]

DATE-ISSUED: August 16, 1932

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
AARON BENOWITZ				

US-CL-CURRENT: 156/63; 156/296, 156/297, 156/72

☐ 25. Document ID: WO 2004028468 A2

L3: Entry 25 of 51 File: EPAB Apr 8, 2004

PUB-NO: WO2004028468A2
DOCUMENT-IDENTIFIER: WO 2004028468 A2
TITLE: METHODS AND COMPOSITIONS FOR TREATMENT OF NEUROLOGICAL DISORDER

PUBN-DATE: April 8, 2004

INVENTOR-INFORMATION:

NAME	COUNTRY
BENOWITZ, LARRY I	US

INT-CL (IPC): A61 K 0/

ABSTRACT:

CHG DATE=20040420 STATUS=O>The present invention provides methods and compositions for producing a neurosalutary effect in a subject useful in treatment of neurological disorders, including retinal and optic nerve damage, in a subject in need thereof. The method includes administering to a subject a therapeutically effective amount of a hexose, such as mannose.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw. Desc

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☐ 26. Document ID: WO 2004001547 A2

L3: Entry 26 of 51 File: EPAB Dec 31, 2003

PUB-NO: WO2004001547A2
DOCUMENT-IDENTIFIER: WO 2004001547 A2
TITLE: TECHNOLOGY ENHANCED COMMUNICATION AUTHORIZATION SYSTEM

PUBN-DATE: December 31, 2003

INVENTOR-INFORMATION:

NAME	COUNTRY
BENOWITZ, JOSEPH C	US
BUNCH, KYLE J	US

INT-CL (IPC): G06 F 0/

ABSTRACT:

CHG DATE=20040128 STATUS=O>A method of authorizing communications includes receiving a communication from a sender (154), determining if the communication contains a valid authorization code (156), notifying the sender (164) if a valid authorization code is not detected with instructions on obtaining a valid authorization code and providing the sender with a service for obtaining a valid authorization code in order to resend the communication with the valid authorization code. The method of authorizing communications also includes forwarding the communication to a recipient if a valid authorization code is detected (158) and holding the communication in an unauthorized box if a valid authorization code is not detected.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw. Desc

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☐ 27. Document ID: WO 3101442 A1

L3: Entry 27 of 51 File: EPAB Dec 11, 2003

PUB-NO: WO003101442A1
DOCUMENT-IDENTIFIER: WO 3101442 A1

TITLE: PEPTIDE DEFORMYLASE INHIBITORS

PUBN-DATE: December 11, 2003

INVENTOR-INFORMATION:

NAME	COUNTRY
AUBART, KELLY M	US
BENOWITZ, ANDREW B	US
CHRISTENSEN, SIEGFRIED B IV	US
KARPINSKI, JOSEPH M	US
LEE, JINHWA	US
SILVA, DOMINGOS J	US

INT-CL (IPC): A61 K 31/16; A61 K 31/18; A61 K 31/44; A61 K 31/50; A61 K 31/505; A61 K 31/495; C07 C 243/22; C07 C 311/16 ; C07 D 213/77; C07 D 237/22; C07 D 239/42
EUR-CL (EPC): C07C243/34; C07C255/66, C07C311/39 , C07D213/77 , C07D215/12 , C07D237/20 , C07D239/42 , C07D239/42 , C07D239/42 , C07D239/48 , C07D239/70 , C07D239/95 , C07D241/44 , C07D251/18 , C07D251/22 , C07D253/08 , C07D307/81 , C07D401/04 , C07D401/12 , C07D403/04 , C07D405/04 , C07D471/04 , C07D473/00 , C07D487/04

ABSTRACT:

CHG DATE=20031223 STATUS=O>Novel PDF inhibitors and novel methods for their use are provided.

☐ 28. Document ID: WO 9911274 A1

L3: Entry 28 of 51 File: EPAB Mar 11, 1999

PUB-NO: WO009911274A1
DOCUMENT-IDENTIFIER: WO 9911274 A1
TITLE: USE OF PURINE NUCLEOSIDES FOR MODULATING THE AXONAL OUTGROWTH OF CENTRAL NERVOUS SYSTEM NEURONS

PUBN-DATE: March 11, 1999

INVENTOR-INFORMATION:

NAME	COUNTRY
BENOWITZ, LARRY I	US

INT-CL (IPC): A61 K 31/70; A61 K 31/52
EUR-CL (EPC): A61K031/52; A61K031/70, A61K031/70

ABSTRACT:

CHG DATE=19990905 STATUS=O>Methods and compositions for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons in conditions such as epilepsy, e.g., post-traumatic epilepsy, and neuropathic pain

syndrome, are also provided. These methods generally involve contacting the central nervous system neurons with a purine nucleoside, or analog thereof. Preferably, inosine or guanosine is used to stimulate axonal outgrowth and 6-thioguanine is used to inhibit axonal outgrowth. The methods and compositions are particularly useful for modulating the axonal outgrowth of mammalian central nervous system neurons, such as mammalian retinal ganglion cells. Pharmaceutical and packaged formulations that include the purine nucleosides, and analogs thereof, of the invention are also provided.

☐ 29. Document ID: WO 9846577 A1

L3: Entry 29 of 51 File: EPAB Oct 22, 1998

PUB-NO: WO009846577A1
DOCUMENT-IDENTIFIER: WO 9846577 A1
TITLE: NOVEL OXAZOLIDINONES AND METHODS FOR THE SYNTHESIS AND USE OF SAME

PUBN-DATE: October 22, 1998

INVENTOR-INFORMATION:

NAME	COUNTRY
SMITH, AMOS B III	
HIRSCHMANN, RALPH F	
SPRENGELER, PAUL A	
BENOWITZ, ANDREW B	
FAVOR, DAVID A	

INT-CL (IPC): C07 D 263/04
EUR-CL (EPC): C07D263/22

ABSTRACT:

CHG DATE=19990905 STATUS=C>Pyrrolinone-based compounds are provided which mimic or inhibit the biological and/or chemical activity of peptides, as are synthetic methods therefor.

☐ 30. Document ID: WO 9606859 A1

L3: Entry 30 of 51 File: EPAB Mar 7, 1996

PUB-NO: WO009606859A1
DOCUMENT-IDENTIFIER: WO 9606859 A1
TITLE: TROPHIC FACTORS FOR CENTRAL NERVOUS SYSTEM REGENERATION

PUBN-DATE: March 7, 1996

INVENTOR-INFORMATION:

NAME COUNTRY

BENOWITZ, LARRY I

IRWIN, CARLEEN A

JACKSON, PAUL

INT-CL (IPC): C07 K 14/475; A61 K 38/00; C07 K 16/22; C12 N 15/12
EUR-CL (EPC): C07K014/475

ABSTRACT:

Cell culture conditions were developed which maintain the nerve cells of the retina in well-defined, serum-free conditions. The molecular factors that stimulate axonal regeneration from these neurons were characterized. The glial sheath cells that surround the axons of the optic nerve release two molecules that trigger and sustain nerve regeneration. One of the molecules is referred to as axogenesis factor 1 (AF-1), and is a low molecular weight polypeptide with a size in the range of 1000 daltons, determined to be about 707 daltons by mass spectroscopy. The second molecule, AF-2, is a larger protein with a size of approximately 12,000 daltons. Studies indicate that these factors are strongly involved in CNS regeneration, and are therefore useful in the treatment of spinal cord and other nervous tissue damage.

☐ 31. Document ID: WO 9408618 A1

L3: Entry 31 of 51 File: EPAB Apr 28, 1994

PUB-NO: WO009408618A1
DOCUMENT-IDENTIFIER: WO 9408618 A1
TITLE: ORAL TOLERANCE AND IMMUNE SUPPRESSION IN THE TREATMENT OF AIDS

PUBN-DATE: April 28, 1994

INVENTOR-INFORMATION:

NAME COUNTRY

BENOWITZ, LARRY I

TRUJILLO, J ROBERTO

IRWIN, CARLEEN A

INT-CL (IPC): A61K 39/12; C12Q 1/68; C07K 3/00
EUR-CL (EPC): C07K014/47

ABSTRACT:

A method of diagnosis and treatment of AIDS-related disorders has been developed based on the presence of an autoimmune response, evoked by infection with the human immunodeficiency virus (HIV), which then leads to the destruction of cells in the immune and nervous systems.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Des

☐ 32. Document ID: AU 2003272728 A1, WO 2004028468 A2

L3: Entry 32 of 51 File: DWPI Apr 19, 2004

DERWENT-ACC-NO: 2004-316013
DERWENT-WEEK: 200462

TITLE: Use of hexose (e.g. D-mannose) to treat/alleviate neurological disorders such as traumatic brain injury, stroke, cerebral aneurysm, Parkinson's disease, amyotrophic lateral sclerosis and Alzheimer's disease

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2002US-414063P (September 27, 2002)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 2003272728 A1	April 19, 2004		000	A61K000/00
WO 2004028468 A2	April 8, 2004	E	059	A61K000/00

INT-CL (IPC): A61 K 0/00

ABSTRACTED-PUB-NO: WO2004028468A
BASIC-ABSTRACT:

NOVELTY - Treatment of a neurological disorder comprises the administration of a hexose (I).

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) an article of manufacture that comprises a pharmaceutical agent (A) (comprising D-mannose) contained within a packaging material which comprises a label indicating that (A) may be administered together with a carrier for a sufficient term at an effective dose to treat a neurological disorder; and

(2) a formulation comprising D-mannose, a cyclic adenosine monophosphate (cAMP) modulator and a carrier.

ACTIVITY - Neuroprotective; Vulnerary; Cerebroprotective; Vasotropic; Antiparkinsonian; Nootropic; CNS-Gen.; Anticonvulsant; Neuroleptic; Muscular-Gen.; Relaxant; Antiinflammatory; Ophthalmological.

The axon-promoting effects of hexose sugars and related compounds were tested on retinal ganglion cells in culture. (I) exhibited a median effective dosage value of approximately 10 mu M.

MECHANISM OF ACTION - None given in the source material.

USE - Treatment with (I) reverses neuronal damage and treats/alleviates neurological disorders (preferably traumatic brain injury, stroke, cerebral aneurysm, Parkinson's disease, amyotrophic lateral sclerosis, Alzheimer's disease, diffuse cerebral cortical atrophy, Lewy-body dementia, Pick disease, mesolimbocortical dementia, thalamic degeneration, Huntington's chorea, cortical-striatal-spinal degeneration, cortical-basal ganglionic degeneration, cerebrocerebellar degeneration, familial dementia with spastic paraparesis, polyglucosan body disease, Shy-Drager syndrome, olivopontocerebellar atrophy, progressive supranuclear palsy, dystonia musculorum deformans, Hallervorden-Spatz disease, Meige syndrome, familial tremors, Gilles de la Tourette syndrome, acanthocytic chorea, Friedreich ataxia, Holmes familial cortical cerebellar atrophy, Gerstmann-Straussler-Scheinker disease, progressive spinal

muscular atrophy, progressive balbar palsy, primary lateral sclerosis, hereditary muscular atrophy, spastic paraplegia, peroneal muscular atrophy, hypertrophic interstitial polyneuropathy, heredopathia atactica polyneuritiformis, optic neuropathy, ophthalmoplegia and, particularly, spinal cord injury (characterized by monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia), retinal damage (resulting from macular degeneration) or optic nerve damage (resulting from glaucoma) (all claimed).

☐ 33. Document ID: AU 2003278421 A1, US 20030236847 A1, WO 2004001547 A2

L3: Entry 33 of 51 File: DWPI Jan 6, 2004

DERWENT-ACC-NO: 2004-131511
DERWENT-WEEK: 200447

TITLE: Communication authorization method for Internet, involves sending notification to sender for obtaining valid authorization code to place e-mail in receiver's authorized in box, when received e-mail does not contain valid code

INVENTOR: BENOWITZ, J C; BUNCH, K J

PRIORITY-DATA: 2002US-390425P (June 19, 2002), 2003US-0465245 (June 19, 2003)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 2003278421 A1	January 6, 2004		000	H04L009/00
US 20030236847 A1	December 25, 2003		031	H04L009/00
WO 2004001547 A2	December 31, 2003	E	000	G06F000/00

INT-CL (IPC): G06 F 0/00; G06 F 11/30; G06 F 15/16; H04 L 9/00; H04 L 9/32

ABSTRACTED-PUB-NO: US20030236847A
BASIC-ABSTRACT:

NOVELTY - The method involves determining whether an e-mail received from a sender contains a valid authorization code. If authorization code is not detected, the e-mail is stored in an unauthorized communication box of a receiver. A notification for obtaining a valid authorization code to place the e-mail in receiver's authorized box, is sent to sender based on the determination result.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) method of preventing e-mail from reaching in box of receiver;

(2) purchase order filling method;

(3) codes usage method;

(4) e-mail distributing method; and

(5) data copying method.

USE - For limiting reception of unwanted e-mails while communicating through Internet. Also applies to instant messaging, peer-to-peer networking, streaming audio, streaming video and any other information transmitted over local and wide area networks, virtual networks and the Internet.

ADVANTAGE - Enables the user to receive only authorized e-mails at any time effectively, by providing authorization code for the e-mails, thereby reception of unsolicited communication is eliminated.

DESCRIPTION OF DRAWING(S) - The figure shows the flowchart explaining communication authorization process.

☐ 34. Document ID: AU 2003247445 A1, WO 2003101442 A1

L3: Entry 34 of 51 File: DWPI Dec 19, 2003

DERWENT-ACC-NO: 2004-108260
DERWENT-WEEK: 200449

TITLE: New N-hydroxy formamide compounds are peptide deformylase inhibitors, useful for the treatment of bacterial infections

INVENTOR: AUBART, K M; BENOWITZ, A B ; CHRISTENSEN, S B ; KARPINSKI, J M ; LEE, J ; SILVA, D J

PRIORITY-DATA: 2002US-384457P (May 31, 2002)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 2003247445 A1	December 19, 2003		000	A61K031/16
WO 2003101442 A1	December 11, 2003	E	139	A61K031/16

INT-CL (IPC): A61 K 31/16; A61 K 31/18; A61 K 31/44; A61 K 31/495; A61 K 31/50; A61 K 31/505; C07 C 243/22; C07 C 311/16 ; C07 D 213/77; C07 D 237/22; C07 D 239/42

ABSTRACTED-PUB-NO: WO2003101442A
BASIC-ABSTRACT:

NOVELTY - N-hydroxy formamide compounds (I) are new.

DETAILED DESCRIPTION - N-hydroxy formamide compounds of formula (I) and their salts, solvates or physiologically functional derivatives are new.

R = 2-6C alkyl, 2-6C alkenyl, 2-6C alkynyl, (CH2)n-3-6C carbocycle (each optionally substituted by alkoxy, halogen or 1-3C alkylsulfanyl) or (CH2)n-R4;

R4 = phenyl, furan, benzodioxane or benzo(1,3)dioxole (all optionally substituted by Cl, Br, I, 1-3C alkyl (optionally substituted by 1-3 F) or 1-2C alkoxy (optionally substituted by 1-3 F));

R1, R2 = H, 1-3C substituted alkyl, 2-3C substituted alkenyl, 2-3C substituted alkynyl, (CH2)n-3-6C substituted carbocycle, aryl, heteroaryl or heterocyclic;

Y = O, CH2 or a covalent bond; and

n = 0-2.

ACTIVITY - Antibacterial.

Test details are described but no results are given.

MECHANISM OF ACTION - Peptide Deformylase Inhibitor.

USE - (I) Are useful for the treatment of bacterial infection (claimed).

ADVANTAGE - The compounds are safe with out any toxicological effects.

☐ 35. Document ID: US 20030153501 A1

L3: Entry 35 of 51 File: DWPI Aug 14, 2003

DERWENT-ACC-NO: 2003-787289
DERWENT-WEEK: 200374

TITLE: Treatment and/or prevention of ocular disorders, e.g. retina or optic nerve damage, comprises administering oncomodulin

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2002US-0294965 (November 14, 2002), 2001US-0872347 (June 1, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 20030153501 A1	August 14, 2003		006	A61K038/17

INT-CL (IPC): A61 K 38/17

ABSTRACTED-PUB-NO: US20030153501A
BASIC-ABSTRACT:

NOVELTY - Ocular disorders are treated and/or prevented by administering oncomodulin.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for an article of manufacture comprising packaging material with a label indicating administration, and oncomodulin.

ACTIVITY - Ophthalmological. No biological data given.

MECHANISM OF ACTION - Adenylate cyclase activator; Macrophage activator; Phosphodiesterase (IV) inhibitor; beta -2 adrenoreceptor inhibitor; beta -2 adrenoreceptor agonist. No biological data given.

USE - The method is used for treating and/or preventing damage to a retina or optic nerve, including damage from ischemic or hypoxic stress, excess intraocular pressure, or injury, in a mammal, e.g. human or nonhuman primate, a dog, a cat, a horse, a cow, or a rodent. It is also useful for treating damage associated with branch and central vein/artery occlusion, angle-closure glaucoma, open-angle glaucoma (claimed), trauma, edema, age related macular degeneration, retinitis pigmentosa, retinal detachments, damage associated with laser therapy (including photodynamic therapy), and surgical light-induced iatrogenic retinopathy.

ADVANTAGE - The invention produces a response or result favorable to the health or function of a neuron, of a part of the nervous system, or of the nervous system generally.

☐ 36. Document ID: US 20020160933 A1

L3: Entry 36 of 51 File: DWPI Oct 31, 2002

DERWENT-ACC-NO: 2003-328371
DERWENT-WEEK: 200331

TITLE: Producing neurosalutary effect, and treating neurological disorder, in a subject, by administering a therapeutically effective amount of a compound that modulates the activity of N-kinase, to the subject

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2001US-0949200 (September 7, 2001), 2000US-0656915 (September 7, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 20020160933 A1	October 31, 2002		020	A61K031/00

INT-CL (IPC): A61 K 31/00

ABSTRACTED-PUB-NO: US20020160933A
BASIC-ABSTRACT:

NOVELTY - Producing (M1) a neurosalutary effect in a subject, and treating a subject suffering from neurological disorder, involves administering a therapeutically effective amount of a compound (I) that modulates the activity of N-kinase, to the subject.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) identifying (M2) a compound capable of producing a neurosalutary effect in a subject, by contacting N-kinase or its biologically active fragment, with a test compound and determining the ability of the test compound to modulate the activity of N-kinase;

(2) a compound capable of producing a neurosalutary effect in a subject identified by the above method;

(3) an isolated N-kinase polypeptide (II) of the type that:

(a) is present in neonatal brain tissue

(b) is inhibited in the presence of 6-thioguanine

(c) is activated in the presence of Mn+2 but not by Mg+2 or Ca+2

(d) has a molecular weight of 49 kDa, and

(e) is eluted from a Cibacron Blue column at a NaCl concentration of 1.5-1.75 M;

(4) an antibody which is specifically reactive with an epitope of (II);

(5) a fragment of (II) comprising at least 15 contiguous amino acids, and capable of eliciting an immune response; and

(6) an isolated nucleic acid molecule (III) encoding a polypeptide comprising a sequence of 272 amino acids fully defined in the specification.

ACTIVITY - Anticonvulsant; Cerebroprotective; Neuroprotective; Nootropic.

No supporting biological data is given.

MECHANISM OF ACTION - Modulator of N-kinase activity (claimed); Promotes neuronal survival, axonal outgrowth and neuronal regeneration; Intracellular mediator of axonal outgrowth.

No supporting biological data is given.

USE - M1 is useful for producing a neurosalutary effect, and thus for treating a subject e.g. mammal, preferably human, suffering from neurological disorder such as spinal cord injury (including monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia), epilepsy, stroke and Alzheimer's disease. The treatment method further involves making a first assessment of a nervous system function prior to administering (I) and making a second assessment of a nervous system function after administering (I) to the subject. The nervous system function is a sensory function, cholinergic innervation or vestibulomotor function (claimed).

(II) is useful as bait protein in a two- or three-hybrid assay, to identify other proteins, which bind to or interact with N-kinase.

☐ 37. Document ID: JP 2004523470 W, WO 200220056 A2, AU 200187118 A, EP 1315514 A2

L3: Entry 37 of 51 File: DWPI Aug 5, 2004

DERWENT-ACC-NO: 2002-393816
DERWENT-WEEK: 200451

TITLE: Producing a neurosalutary effect in a subject e.g., one suffering from neurological disorder such as stroke, to treat the subject, by administering a compound that modulates activity of N-kinase

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2000US-0656915 (September 7, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2004523470 W	August 5, 2004		077	A61K045/00
WO 200220056 A2	March 14, 2002	E	042	A61K045/00
AU 200187118 A	March 22, 2002		000	A61K045/00
EP 1315514 A2	June 4, 2003	E	000	A61K038/18

INT-CL (IPC): A61 K 9/10; A61 K 9/127; A61 K 38/18; A61 K 45/00; A61 P 9/10; A61 P 9/12; A61 P 25/00; A61 P 25/02; A61 P 25/08; A61 P 25/14; A61 P 25/16; A61 P 25/18; A61 P 25/24; A61 P 25/28; A61 P 43/00; C07 K 14/475; C07 K 16/40; C12 N 9/12; C12 N 15/09; C12 Q 1/48; G01 N 33/15; G01 N 33/50; G01 N 33/53; G01 N 33/566

ABSTRACTED-PUB-NO: WO 200220056A
BASIC-ABSTRACT:

NOVELTY - Producing (M1) a neurosalutary effect in a subject e.g., a subject suffering from a neurological disorder, to treat the subject suffering from the neurological disorder, involving administering to the subject a compound (I) that modulates the activity of N-kinase, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated N-kinase polypeptide (II) of the type that: is present in neonatal brain tissue; is inhibited in the presence of 6-thioguanine; is activated in the presence of Mn2+, but not by Mg2+ or Ca2+; has a molecular weight of approximately 49 kDa; and is eluted from a Cibacron Blue column at a sodium chloride concentration of 1.5-1.75 M;

(2) an antibody (III) which is specifically reactive with an epitope of (II);

(3) a fragment (IV) of (I), which comprises at least 15 contiguous amino acids, and is able to elicit an immune response;

(4) an isolated nucleic acid molecule that encodes (II); and

(5) a compound capable of producing a neurosalutary effect in a subject identified using (II).

ACTIVITY - Nootropic; neuroprotective; cerebroprotective; anticonvulsant; vulnerary; tranquilizer; antiparkinsonian; antimanic; antidepressant.

MECHANISM OF ACTION - N-kinase activity modulator; neuronal survival modulator; neuronal regeneration modulator; neuronal axonal outgrowth of central nervous system neurons e.g., retinal ganglion cells, modulator (all claimed).

No data given.

USE - (I) is useful for producing a neurosalutary effect in a subject e.g., a subject suffering from a neurological disorder, to treat the subject (preferably, humans) suffering from the neurological disorder. The neurosalutary effect is produced by modulating neuronal survival, modulating neuronal regeneration or modulating neuronal axonal outgrowth of central nervous system neurons e.g., retinal ganglion cells, in a subject suffering from a neurological disorder such as spinal cord injury characterized by monoplegia, diplegia, paraplegia, hemoplegia and quadriplegia, or suffering from epilepsy, stroke or Alzheimer's disease.

(II) is useful for identifying a compound capable of producing a neurosalutary effect in a subject, preferably a compound which inhibits or stimulates the activity of N-kinase, which involves contacting (II) or its biologically active fragment with a test compound and determining the ability of the test compound to modulate the activity of N-kinase, thereby identifying a compound capable of producing a neurosalutary effect in a subject. The ability of the test compound to modulate the activity of N-kinase is determined by assessing the ability of the test compound to modulate N-kinase-dependant phosphorylation of a substrate. Optionally, (I) is identified using (II) by the following method which involves contacting (II) or its biologically active fragment, with a test compound, an N-kinase substrate (e.g., histone HF-1 protein), radioactive ATP (preferably gamma -32P), and Mn2+; and determining the ability of the test compound to modulate N-kinase dependent phosphorylation of the substrate, thereby identifying a compound capable of producing a neurosalutary effect in a subject. (II) used in the methods described above is preferably a recombinantly produced human N-kinase. Optionally, (II) is bovine N-kinase purified from a bovine source. The methods further involve determining the ability of the test compound to modulate axonal outgrowth of central nervous system neuron (all claimed).

(M1) is useful for treating a neurological disorder such as dementia's related to Alzheimer's disease, Parkinson's disease, senile dementia, Huntington's disease,

Creutzfeldt-Jakob disease, Korsakoff's psychosis, mania, anxiety disorders, obsessive-compulsive disorder, anxiety, bipolar affective disorder. The methods are useful for preventing or treating neurological deficits in embryos or fetuses in utero, in premature infants, or in children with need of such treatment, including those with neurological birth defects. (I) is also useful for modulating activity of N-kinase, in vitro to modulate axonal outgrowth in vitro.

☐ 38. Document ID: WO 200206341 A1, AU 200180566 A

L3: Entry 38 of 51 File: DWPI Jan 24, 2002

DERWENT-ACC-NO: 2002-291790
DERWENT-WEEK: 200236

TITLE: Isolated neurotrophic factor useful for treating neurological conditions is present in a medium containing Schwann cells culture

INVENTOR: BENOWITZ, L I; IRWIN, C A ; JACKSON, P

PRIORITY-DATA: 2000US-0616287 (July 14, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200206341 A1	January 24, 2002	E	050	C07K014/47
AU 200180566 A	January 30, 2002		000	C07K014/47

INT-CL (IPC): C07 K 14/47

ABSTRACTED-PUB-NO: WO 200206341A
BASIC-ABSTRACT:

NOVELTY - An isolated neurotrophic factor (I) of the type that is present in a medium containing Schwann cells culture is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) a method for producing a neurosalutary effect in a subject by administering the neurotrophic factor (I) to the subject; and

(2) a method for treating neurological disorder involving administering (I) to a subject suffering from the neurological disorder.

ACTIVITY - Anticonvulsant; Nootropic; Neuroprotective; Cerebroprotective; Tranquilizer; Vulnerary; Neuroleptic; Antidepressant; Antidiabetic; Antiparkinsonian; Antimanic; Hypotensive; Analgesic; Antibacterial; Antiinflammatory; Antipyretic; Anti-HIV.

MECHANISM OF ACTION - Axonal outgrowth of naive goldfish retinal ganglion cells stimulator; Axonal outgrowth of embryonic rat spinal cord neuron stimulator; Modulators of neuronal survival, neuronal regeneration and neuronal axonal outgrowth of central nervous system neurons such as retinal ganglion cells.

USE - For producing neurosalutory effect in a subject such as mammal e.g. human suffering from a neurological disorder such as spinal cord injury, e.g. monoplegia, diplegia, paraplegia, hemiplegia, or quadriplegia, epilepsy e.g. posttraumatic epilepsy, Alzheimer's disease (all claimed). The neurological disorders include

traumatic or toxic injuries to peripheral or cranial nerves, traumatic brain injury, stroke, cerebral aneurism, cognitive and neurodegenerative disorders such as dementias, Huntington's disease, Gilles de la Tourette's syndrome, multiple sclerosis, amyotrophic, lateral sclerosis, hereditary motor and sensory neuropathy (Charcot-Marie-Tooth disease), diabetic neuropathy, progressive supranuclear palsy, Jakob-Creutzfeldt disease or disorders included in Harrison's Principles of Internal Medicine (Braunwald et-al.McGraw-Hill,2001) and in the American Psychiatric Association's Diagmestic and statistical manual of mental Disorders DSM-IV (American Psychiatric Press,2000); for treating hypertension and sleep disorders, neuropsychiatric disorders such as depression, schizophrenia, schizoaffective disorder, korsakoff's psychosis, mania, anxiety disorders, or phobic disorder, learning or memory disorders (such as amnesia and age-related memory loss), attention deficit disorder, dysthymic disorder, major depressive disorder, mania, obsessive compulsive disorder, psychoactive substance, use disorder, panic disorder, bipolar affective disorder, psychogenic pain syndromes, and eating disorders; for treating injuries of nervous system due to an infections disease (such as meningitis, high fever of various etiologies, HIV, syphilis, or post-polio syndrome) or due to electricity (including contact with electricity or lightening and complications from electro-convulsive psychiatric therapy); for preventing or treating neurological deficits in embryos or fetuses in utero, in premature infants, or in children with need of such treatment, including those with neurological birth defects.

ADVANTAGE - The formulation provides sustained delivery of (I) for at least one-week (preferably at least one month) after the formulation is administered to the subject. The neurotrophic factor stimulates axonal outgrowth of naive goldfish retinal ganglion cells, embryonic rat spinal cord neurons and passes through a centrifugal filter with a 1 kDa cut-off. The neurotrophic factor further fails to bind to a 18C reversed-phase HPLC column, forms a compound that elutes from a reverse-phase HPLC column, at 23 minutes, after being chemically derivatized with AQC and has an elution time of 6 minutes on a G10-Sepharose size-exclusive column.

☐ 39. Document ID: JP 2003534385 W, WO 200191783 A2, AU 200168147 A, US 20020119923 A1, EP 1289540 A2

L3: Entry 39 of 51 File: DWPI Nov 18, 2003

DERWENT-ACC-NO: 2002-097736
DERWENT-WEEK: 200401

TITLE: Method of producing a neurosalutary effect in a subject with a neurological condition, comprises administering a macrophage derived factor

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2000US-208778P (June 1, 2000), 2001US-0872347 (June 1, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2003534385 W	November 18, 2003		045	A61K035/26
WO 200191783 A2	December 6, 2001	E	035	A61K038/18
AU 200168147 A	December 11, 2001		000	A61K038/18
US 20020119923 A1	August 29, 2002		000	A61K038/18
EP 1289540 A2	March 12, 2003	E	000	A61K038/18

INT-CL (IPC): A61 K 9/10; A61 K 9/12; A61 K 9/127; A61 K 31/00; A61 K 31/45; A61 K

31/7076; A61 K 31/7105; A61 K 31:00; A61 K 35/26; A61 K 38/18; A61 K 38/22; A61 K 38:18; A61 K 45/00; A61 P 25/00; A61 P 25/08; A61 P 25/28; A61 P 43/00; A61 K 38/18; A61 K 31:00

ABSTRACTED-PUB-NO: US20020119923A
BASIC-ABSTRACT:

NOVELTY - A method of producing a neurosalutary effect in a subject with a neurological condition comprises administering a macrophage-derived factor.

DETAILED DESCRIPTION - A method of producing a neurosalutary effect in a subject with a neurological condition comprises administering a macrophage-derived factor, and optionally a cAMP modulator or an axogenic factor. INDEPENDENT CLAIMS are included for:

(1) compositions for producing a neurosalutary effect comprising a macrophage-derived factor and optionally a cAMP modulator or an axogenic factor;

(2) a method comprising the administration of oncomodulin, and for producing a neurosalutary effect with an effective amount of AF-1; and

(3) a composition comprising macrophage-derived factor and carrier packed with instructions for use of a pharmaceutical composition.

ACTIVITY - Anticonvulsant; nootropic; neuroprotective; antiparkinsonian; nootropic; anticonvulsant; neuroleptic; antidiabetic; antidepressant; tranquilizer.

No specific biological data given.

MECHANISM OF ACTION - Neuronal survival modulator; neuronal regeneration modulator; neuronal axonal outgrowth modulator.

USE - For treating neurological disorders, e.g. spinal cord injury, such as monoplegia, diplegia, paraplegia, hemiplegia or quadriplegia; epilepsy, such as posttraumatic epilepsy; or Alzheimer's disease (claimed), also Parkinson's disease, senile dementia, Huntington's disease, Gilles de la Tourette's syndrome, multiple sclerosis, amyotrophic lateral sclerosis, hereditary motor and sensory neuropathy, diabetic neuropathy, progressive supranuclear palsy, Creutzfeldt-Jakob disease, depression, schizophrenia, schizoaffective disorder, Korsakoff's psychosis, mania, anxiety disorders, phobic disorders, learning or memory disorders, attention deficit disorder, dysthymic disorder, major depressive disorder, mania, obsessive-compulsive disorder, psychoactive substance use disorder, anxiety, phobias, panic disorder, bipolar affective disorder, psychogenic pain syndromes and eating disorders.
ABSTRACTED-PUB-NO:

WO 200191783A EQUIVALENT-ABSTRACTS:

NOVELTY - A method of producing a neurosalutary effect in a subject with a neurological condition comprises administering a macrophage-derived factor.

DETAILED DESCRIPTION - A method of producing a neurosalutary effect in a subject with a neurological condition comprises administering a macrophage-derived factor, and optionally a cAMP modulator or an axogenic factor. INDEPENDENT CLAIMS are included for:

(1) compositions for producing a neurosalutary effect comprising a macrophage-derived factor and optionally a cAMP modulator or an axogenic factor;

(2) a method comprising the administration of oncomodulin, and for producing a neurosalutary effect with an effective amount of AF-1; and

(3) a composition comprising macrophage-derived factor and carrier packed with instructions for use of a pharmaceutical composition.

ACTIVITY - Anticonvulsant; nootropic; neuroprotective; antiparkinsonian; nootropic; anticonvulsant; neuroleptic; antidiabetic; antidepressant; tranquilizer.

No specific biological data given.

MECHANISM OF ACTION - Neuronal survival modulator; neuronal regeneration modulator; neuronal axonal outgrowth modulator.

USE - For treating neurological disorders, e.g. spinal cord injury, such as monoplegia, diplegia, paraplegia, hemiplegia or quadriplegia; epilepsy, such as posttraumatic epilepsy; or Alzheimer's disease (claimed), also Parkinson's disease, senile dementia, Huntington's disease, Gilles de la Tourette's syndrome, multiple sclerosis, amyotrophic lateral sclerosis, hereditary motor and sensory neuropathy, diabetic neuropathy, progressive supranuclear palsy, Creutzfeldt-Jakob disease, depression, schizophrenia, schizoaffective disorder, Korsakoff's psychosis, mania, anxiety disorders, phobic disorders, learning or memory disorders, attention deficit disorder, dysthymic disorder, major depressive disorder, mania, obsessive-compulsive disorder, psychoactive substance use disorder, anxiety, phobias, panic disorder, bipolar affective disorder, psychogenic pain syndromes and eating disorders.

☐ 40. Document ID: EP 1466606 A2, WO 9911274 A1, AU 9866568 A, EP 1009412 A1, CN 1286632 A, KR 2001023578 A, JP 2001516695 W, US 20020042390 A1, US 20020055484 A1, AU 748961 B, US 6440455 B1, US 20020128223 A1, US 20020137721 A1, NZ 503073 A, US 6551612 B2, RU 2212241 C2, US 20040014710 A1, EP 1009412 B1, DE 69825292 E

L3: Entry 40 of 51 File: DWPI Oct 13, 2004

DERWENT-ACC-NO: 1999-228934
DERWENT-WEEK: 200467

TITLE: Modulating axonal outgrowth of central nervous system

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 1997US-0921902 (September 2, 1997), 2001US-0997688 (November 29, 2001), 2001US-0997687 (November 29, 2001), 2002US-0145224 (May 14, 2002), 2002US-0144952 (May 14, 2002), 2003US-0385031 (March 10, 2003)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 1466606 A2	October 13, 2004	E	000	A61K031/52
WO 9911274 A1	March 11, 1999	E	043	A61K031/70
AU 9866568 A	March 22, 1999		000	
EP 1009412 A1	June 21, 2000	E	000	
CN 1286632 A	March 7, 2001		000	A61K031/70
KR 2001023578 A	March 26, 2001		000	A61K031/70
JP 2001516695 W	October 2, 2001		047	A61K031/708
US 20020042390 A1	April 11, 2002		000	A61K031/708
US 20020055484 A1	May 9, 2002		000	A61K031/7105
AU 748961 B	June 13, 2002		000	A61K031/70
US 6440455 B1	August 27, 2002		000	A61K009/127
US 20020128223 A1	September 12, 2002		000	A61K031/708

US 20020137721 A1	September 26, 2002		000	A61K031/708
NZ 503073 A	November 22, 2002		000	A61K031/70
US 6551612 B2	April 22, 2003		000	A61K009/127
RU 2212241 C2	September 20, 2003		000	A61K031/70
US 20040014710 A1	January 22, 2004		000	A61K031/7076
EP 1009412 B1	July 28, 2004	E	000	A61K031/70
DE 69825292 E	September 2, 2004		000	A61K031/70

6551612 B2 , RU 2212241 C2 , US 20040014710 A1 INT-CL (IPC): A61 K 9/127; A61 K 9/16; A61 K 31/52; A61 K 31/522; A61 K 31/70; A61 K 31/7076; A61 K 31/708; A61 K 31/7105; A61 P 25/00; A61 P 25/08; A61 P 43/00; C07 H 19/167

ABSTRACTED-PUB-NO: US 6440455B
BASIC-ABSTRACT:

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.
ABSTRACTED-PUB-NO:

US20020042390A EQUIVALENT-ABSTRACTS:

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50

values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.

US20020055484A

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.

US20020128223A

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or inhibiting the outgrowth. The method is particularly useful for stimulating axonal

outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.

US20020137721A

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.

WO 9911274A

☐ 41. Document ID: WO 9846577 A1, US 6034247 A

L3: Entry 41 of 51 File: DWPI Oct 22, 1998

DERWENT-ACC-NO: 1998-583263
DERWENT-WEEK: 200019

TITLE: New 4-alkenyl-5-oxazolidinone compounds - used in preparation of pyrrolinone based peptide analogues which mimic or inhibit natural peptide activity

INVENTOR: BENOWITZ, A B; FAVOR, D A ; HIRSCHMANN, R F ; SMITH, A B ; SPRENGELER, P A

PRIORITY-DATA: 1997US-0843031 (April 11, 1997), 1993US-0018696 (February 17, 1993), 1994US-0285027 (August 2, 1994)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9846577 A1	October 22, 1998	E	058	C07D263/04
US 6034247 A	March 7, 2000		000	C07D263/04

INT-CL (IPC): C07 D 263/04

ABSTRACTED-PUB-NO: US 6034247A
BASIC-ABSTRACT:

4-Alkenyl-5-oxazolidinone compounds of formula (II; Q = H) are new. A claimed synthetic method for a broader range of compounds of formula (II) involves: (a) reacting an aminoacid of formula (I) with an aldehyde of formula R2CHO and a halo compound (or analogue) of formula R3-X in presence of a base to give (II;Q = H); and optionally (b) reacting the product with either (i) a halo compound (or analogue) of formula R6-Z in presence of base to give (II; Q = R6) or (ii) bromoacetonitrile or acrylonitrile in presence of base to give (II; Q = R4), optionally followed by reaction with aqueous acid to give (II; Q = R'4). R1 = 1-10C alkyl; R2 = 1-10C alkyl or 3-10C aryl; R3 = amine protecting group; R4 = CH2CN or CH2CH2CN; R'4 = side-chain of natural or non-natural asparagine or glutamine; R6 = side-chain of a natural or non-natural aminoacid; X,Z = halo or other leaving group. Another claimed synthetic method involves reaction of (II; Q = R6) with a base (preferably potassium hydroxide) to give an aminoacid ester of formula (VI; R'3 = R3) and optionally converting the product into the free acid (VI; R'3 = H) in the presence of a catalyst.

USE - (II) and (VI) are used in the preparation of pyrrolinone compounds for use in place of aminoacids in peptide compounds. The peptide analogues containing pyrrolinone units mimic or inhibit the biological and/or chemical activity of natural peptides, are more stable than the peptides and can assume the conformation of a beta -pleated peptide strand. The peptide analogues specifically function as enzyme inhibitors, e.g. as renin inhibitors, serine or cysteine protease inhibitors or HIV-1 inhibitors. See also US5489692.
ABSTRACTED-PUB-NO:

WO 9846577A EQUIVALENT-ABSTRACTS:

4-Alkenyl-5-oxazolidinone compounds of formula (II; Q = H) are new. A claimed synthetic method for a broader range of compounds of formula (II) involves: (a) reacting an aminoacid of formula (I) with an aldehyde of formula R2CHO and a halo compound (or analogue) of formula R3-X in presence of a base to give (II;Q = H); and optionally (b) reacting the product with either (i) a halo compound (or analogue) of formula R6-Z in presence of base to give (II; Q = R6) or (ii) bromoacetonitrile or acrylonitrile in presence of base to give (II; Q = R4), optionally followed by reaction with aqueous acid to give (II; Q = R'4). R1 = 1-10C alkyl; R2 = 1-10C alkyl or 3-10C aryl; R3 = amine protecting group; R4 = CH2CN or CH2CH2CN; R'4 = side-chain of natural or non-natural asparagine or glutamine; R6 = side-chain of a natural or non-natural aminoacid; X,Z = halo or other leaving group. Another claimed synthetic method involves reaction of (II; Q = R6) with a base (preferably potassium hydroxide) to give an aminoacid ester of formula (VI; R'3 = R3) and optionally converting the

product into the free acid (VI; R'3 = H) in the presence of a catalyst.

USE - (II) and (VI) are used in the preparation of pyrrolinone compounds for use in place of aminoacids in peptide compounds. The peptide analogues containing pyrrolinone units mimic or inhibit the biological and/or chemical activity of natural peptides, are more stable than the peptides and can assume the conformation of a beta -pleated peptide strand. The peptide analogues specifically function as enzyme inhibitors, e.g. as renin inhibitors, serine or cysteine protease inhibitors or HIV-1 inhibitors. See also US5489692.

☐ 42. Document ID: US 5534849 A

L3: Entry 42 of 51 File: DWPI Jul 9, 1996

DERWENT-ACC-NO: 1996-333400
DERWENT-WEEK: 199633

TITLE: Magnetically actuated switch system having reduced sensitivity to false alarms and reduced power consumption - has signal processing circuit controlled by microprocessor that selectively enables magnetic sensors, and compares corresp. sensor outputs to automatically calibrated level

INVENTOR: BENOWITZ, M; MCDONALD, K B ; PIERCE, S W ; TERRETT, D S

PRIORITY-DATA: 1993US-0105588 (August 11, 1993), 1994US-0351566 (December 6, 1994)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 5534849 A	July 9, 1996		015	G08B013/08

INT-CL (IPC): G08 B 13/08

ABSTRACTED-PUB-NO: US 5534849A
BASIC-ABSTRACT:

The system for detecting a relative placement of moveable and fixed members of a barrier outside of a spatial window defined by minimum and maximum allowable distances between the moveable and fixed members, comprises a magnetic field source to provide a multi-directional proximal magnetic field.

Multiple determinably selectable magnetic field sensors produce output signals responsive to the proximal magnetic field.

A sensor output signal responsive electrical circuit selects fewer than all of the determinably selectable sensors at a time and selectively establishes first and second retainable sensor output signal levels corresp. to the respective minimum and maximum allowable distances between the moveable and fixed members that define the spatial window outside of which relative placement of the moveable and fixed barrier members enables the sensor output signal responsive electrical circuit to notify a monitoring system.

USE/ADVANTAGE - E.g. for physical security monitoring systems. Greater resistance to physical and magnetic tampering.

☐ 43. Document ID: WO 9606859 A1, CN 1164858 A, AU 9535393 A, EP 777686 A1, NZ 293048 A, JP 10505238 W, KR 97705577 A, US 5898066 A, AU 713028 B, AU 200012463 A, RU 2157223 C2

L3: Entry 43 of 51 File: DWPI Mar 7, 1996

DERWENT-ACC-NO: 1996-160307
DERWENT-WEEK: 200148

TITLE: Isolated neurotrophic polypeptide(s) - useful for inducing axonal extension in neuronal cells, partic. for treating nervous tissue damage

INVENTOR: BENOWITZ, L I; IRWIN, C A ; JACKSON, P

PRIORITY-DATA: 1994US-0296661 (August 26, 1994), 2000AU-0012463 (January 18, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9606859 A1	March 7, 1996	E	076	C07K014/475
CN 1164858 A	November 12, 1997		000	C07K014/475
AU 9535393 A	March 22, 1996		000	C07K014/475
EP 777686 A1	June 11, 1997	E	000	C07K014/475
NZ 293048 A	January 26, 1998		000	C07K014/475
JP 10505238 W	May 26, 1998		062	C12N015/09
KR 97705577 A	October 9, 1997		000	C07K014/475
US 5898066 A	April 27, 1999		000	C07K002/00
AU 713028 B	November 18, 1999		000	C07K014/475
AU 200012463 A	July 6, 2000		000	C07K007/06
RU 2157223 C2	October 10, 2000		000	A61K035/60

INT-CL (IPC): A61 K 35/60; A61 K 38/00; A61 K 38/22; C07 K 2/00; C07 K 7/06; C07 K 7/08; C07 K 14/435; C07 K 14/475; C07 K 14/48; C07 K 14/52; C07 K 16/22; C12 N 15/09; C12 N 15/12; C12 P 21/02; C12 P 21/08; C12 Q 1/68

ABSTRACTED-PUB-NO: US 5898066A
BASIC-ABSTRACT:

(A) A neurotrophic polypeptide (NP) is claimed which is selected from: (a) a polypeptide having a mol.wt. of about 700 D by mass spectroscopy, which can be isolated from medium in which glial sheath cells have been cultured by mol. wt. sepn., two-phase extn. and reversed phase HPLC, which retains activity after heating at 95 deg.C for < 15 mins., which retains activity after digestion with pronase and trypsin, and is hydrophilic; and (b) a protein having a mol.wt. of about 12 000 D which can be isolated from medium in which glial sheath cells have been cultured by mol.wt. sepn. and anion exchange chromatography, where the protein binds to the column at pH 10 but not at pH 8.4 and can be eluted with 0.2 M NaCl, loses activity upon heating at 95 deg.C for 15 mins. and loses activity following digestion with trypsin or proteinase K. Also claimed are: (B) a nucleotide sequence encoding a NP as in (A); and (C) an antibody immunoreactive with a NP as in (A).

USE - The NPs can be used for inducing axonal extension in neuronal cells (claimed). They can be used in the treatment of optic nerve, brain, spinal cord and other nervous tissue damage. They can be used for treating e.g. trauma damage, demyelinating diseases, autoimmune disorders or degenerative diseases. The prods. can also be used for the screening of drugs which modulate the activity and/or the

expression of the NPs and in screening of patient samples for the presence of functional NPs.
ABSTRACTED-PUB-NO:

WO 9606859A EQUIVALENT-ABSTRACTS:

(A) A neurotrophic polypeptide (NP) is claimed which is selected from: (a) a polypeptide having a mol.wt. of about 700 D by mass spectroscopy, which can be isolated from medium in which glial sheath cells have been cultured by mol. wt. sepn., two-phase extn. and reversed phase HPLC, which retains activity after heating at 95 deg. C for < 15 mins., which retains activity after digestion with pronase and trypsin, and is hydrophilic; and (b) a protein having a mol.wt. of about 12 000 D which can be isolated from medium in which glial sheath cells have been cultured by mol.wt. sepn. and anion exchange chromatography, where the protein binds to the column at pH 10 but not at pH 8.4 and can be eluted with 0.2 M NaCl, loses activity upon heating at 95 deg. C for 15 mins. and loses activity following digestion with trypsin or proteinase K. Also claimed are: (B) a nucleotide sequence encoding a NP as in (A); and (C) an antibody immunoreactive with a NP as in (A).

USE - The NPs can be used for inducing axonal extension in neuronal cells (claimed). They can be used in the treatment of optic nerve, brain, spinal cord and other nervous tissue damage. They can be used for treating e.g. trauma damage, demyelinating diseases, autoimmune disorders or degenerative diseases. The prods. can also be used for the screening of drugs which modulate the activity and/or the expression of the NPs and in screening of patient samples for the presence of functional NPs.

☐ 44. Document ID: WO 9408618 A1, AU 9453590 A

L3: Entry 44 of 51 File: DWPI Apr 28, 1994

DERWENT-ACC-NO: 1994-150948
DERWENT-WEEK: 199817

TITLE: Diagnosis and treatment of AIDS related disorders, esp. AIDS related dementia - using antibodies raised by an autoimmune responsive caused by infection with HIV.

INVENTOR: BENOWITZ, L I; IRWIN, C A ; TRUJILLO, J R

PRIORITY-DATA: 1992US-0959771 (October 13, 1992)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9408618 A1	April 28, 1994	E	049	A61K039/12
AU 9453590 A	May 9, 1994		000	A61K039/12

INT-CL (IPC): A61K 39/12; C07K 3/00; C12Q 1/68

ABSTRACTED-PUB-NO: WO 9408618A
BASIC-ABSTRACT:

A novel method (I) for treating AIDS related disorders comprises: (a) determining in a patient sample the presence of human proteins immuno-reactive with anti-HIV antibodies; (b) isolating peptides from the immunoreactive proteins which bind to the anti-HIV antibodies (Abs); and (c) admin. an effective amt. of the immunoreactive peptides to the patient to induce tolerance to the protein; and opt. further boosting

immunity to HIV-1 by presenting systemically recombinant mols. of the AIDS viral coat that do not resemble portions of gp120 or gp41.

USE - Some elements of AIDS result from an autoimmune reaction occurring in patients infected with the virus. The presence of the anti-HIV Abs that cross react with normal human proteins is a diagnostic predictor of patients who may develop AIDS related dementia and other aspects of AIDS related disorders. The cDNA encoding the cross-reactive portions of the protein, or the protein itself can be used to make peptides for induction of tolerance in AIDS patients. Immunologically cross-reactive proteins are found on other tissues.

☐ 45. Document ID: US 3466397 A

L3: Entry 45 of 51 File: USOC Sep 9, 1969

US-PAT-NO: 3466397
DOCUMENT-IDENTIFIER: US 3466397 A

TITLE: CHARACTER AT A TIME DATA MULTIPLEXING SYSTEM

DATE-ISSUED: September 9, 1969

US-CL-CURRENT: 370/479; 370/246, 370/501, 370/503, 370/522

DOCUMENT TEXT:

Sept. 9, 1969 P. BENOWITZ ET AL 3,466,397 CHARACTER AT A TIME DKTA MULTIPLEXING SYSTEM Filed Dec. 14, 1965 6 Sheets-Sheet 1 C L CL F- F- F- -D :D :D C\i 0 0 0 0 < < < t@ < 0 0 - C\i z F- z F- rr Cl- F- (:I_ :D cr =) r-r- U) a- :D (/) CL LL, F- LLJ F- cr_ LLJ F- F- L.L f') LLi Li- (.0 :D LL (f) @- U- LL (f) Li- => L.L Li. O:D 0 o=) O:D co co rn co (f) j C:D T cl-c,\j < Z F- :D 0 0 F- 2 z 0 LO C\i L) C) C\i 0 Z z < -00 CL CL. u u < < @-- C\J < :D o 0 < < cr F z F-cc Q- :DLL, LLJ F- C) LLJ F- 0- F- 0 LL LL _Uj _U_ ZLI- -LL ZLL Z u- 7 - L L co co :D co co co C) C) C:) F- F- => Q- n. Z z C\i < C:) < < no n P BENO W/ rz lAtVEAt TOqS M. rONAT OWIrz 0. M. R(ITEL M N BY A T T O P N E Y

Sept. 9, 1969 314669397 P. BENOWITZ ET AL CHARACTER AT A TIME DATA MULTIPLEXING SYSTEM Filed Dec. 14, 1965 6 Sheets-Sheet 2 C"i 0) cn I C\j C'\i C \ i r) co w C\J rj cr_ r\.l M C\4 :z CD C\i Ln ---------- rq ------ cr L LL U) C'4 LL CD C \i Z) 2 (D Ln rf) co Ln U-) C\J. (\i L.L CD (D OC) U-) C\J C\) LO C.L U') Z C\i 0 00 C> U') C\l C\i CD C\J C \l C\i C\4 < z

Sept. 9, 1969 P. BENOWITZ ET AL 3,466,397 CHARACTER AT A TIME DATA MULTIPLEXING SYSTEM Filed Dec. 14, 1965 6 Sheets-Sheet 3 IA)01:) 01 "D]NOD 'V40D Oi co O. co r \ j co C) " rl- C\i rn \i CQ C\i C\J- m to C\i cn C\i C\i C> O D C\i C=) ,@ml C\@ C\i I (n ui CD 'co C,\Oj rn Zt,Zt--@- u c@, en ,\j C\i C) rn C, C\i co C\i C\i

Sept. 9, 1969' P. IBENOWITZ ET AL 3,466@397 CHARACTER AT A TIME DATA MULTIPLEXING SYSTEWL Filed Dec. 14, 1965 @6 Sheets@-Sh@@bt 4 U) OM C\i CO c@ U-) Ln cc C) 0 C) C:) m , 0 OD u CL m Ln 0 LC) u LO Ln 0 Lr) (D U. C) C\i C) F- C\i C\i C). C :) , C) .co C\i C:) C:) z cuo) U 0 0 --T I z C :) (Z) 7 C\i z Z I - 4 @- 4 @lcl_ 4 co C13 co LA- z

Sept. 9, 1969 P. BF- NOWITZ ET AL 314669397 CHARACTER AT A TIME DATA MULTIPLEXING SYSTEM Filed Dec. 14, 1965 6 Sheets-Sheet 5 C:) LO Ln u) C\i lLj aD C) L C) CQ LC) LO LO 0- c_@ cm LO Ln to ro LO C\i to LO Ln cn LO LO C\j LO Lf) (.0 f 400 cn, te) CC co Lr) C> Ln C\j LU Z U- o 7 Cf3 @ui C> T- LI) tc) CO LO Lr) ui z m z c:l Lr CL 0 LO LO LO 0 LO co LO LO CL ri @0-1 C 14) Ca VY c o cci F- :z

Sept. 9, 1969 P. BENOWITZ ET AL 3t466,397 CHARACTER AT A TIME DATA MULTIPLEXING SYSTEM Filed Dec.'14, 1965 6 Sheets-Sheet 6 C\i :D 0 LC) C\i I-Z < CO C) C\J C\J C\i Ln C) " CO C> C\i (.0 ro LO a. co U:z (00 OU LO rn 10 LO C, C\J C" m LO C) Ln tn C\i c:@ u-) ko LO co, CL iz V) LO U-1 LO c:o 1* ul)

-United States Patent Office 3@466@397 3,466,397 CHARACTER AT A TIME DATA MULTIPLEXING SYSTEM Paul Benowitz, Brooklyn, Michael Ignatowitz, Flushing, and David M. Tutelman, Bronx, N.Y., assignors to Bell Telephone Laboratories, Incorporated, New York, N.Y., a corporation of New York Filed Dec. 141, 1965, Ser. No. 513,742 Int. Cl. H04j 3102 U.S. Cl. 179-15 13 Claims ABSTRACT OF THE DISCLOSURE Each input port of a data multiplexing system applies a character at a time rather than a bit at a time to the common highway. After transmitting the character bits, the input port enables the next subsequent port whereby various codes and speeds may be mixed. At the other end of the highway, each output port registers the character from the input port and thereupon enables the next subsequent output port to register the character from its corresponding input port. The receiving end includes equipment for detecting loss of synchronization and for thereupon precluding bit registration by the output ports until synchronization is recovered. A Rag bit indicates parity of the character elements and alternatively distinguishes ',)it sequences for "idle" or "break@' conditions from character bits. This invention relates to a system for transmitting data characters and, more particularly, to a data transmission system for interconnecting incoming signalling lines with corresponding outgoing lines by way of a common transmission path or bus on a time division basis. . It is a broad object of this invention to provide an improved time division multiplex data transmission system. When a plurality of data signaling channels are handled by a common facility, it is sometimes convenient tO multiplex the signals from all the channels on a common path on a time division basis. Each incoming line applies the signals to an input port of the multiplex system. The input ports are sequentially scanned and a data bit from each input port is.transmitted to a conimon bus together with a framing or synchronizing signal during each scan cycle. At the remote terminal the data bits from the bus are distributed under control of the framing signal to output ports corresponding to the input ports. Each output port applies the distributed bits to an associated outgoing line reconstructing the signals received from the associated incoming line. Multiplex systems transmitting a bit at a time from each input port during each scan cycle are highly efficient when all individual lines are dedicated to the same data code at identical signaling rates. These systems, however, are not readily adaptable to arrangements where signaling lines are dedicated to different codes and signaling rates. In addition, since multiplex systems transmit the data bits of the code characters interleaved with data bits of other channels, communication of line conditions, such as "idle" or "break@' conditions, in the absence of code signaling, is difficult to accomplish. Systems of this type are also highly dependent on the maintenance of synchronization since the loss thereof results in the improper distribution of the data bits to the output ports thus transmitting intelligence data to the wrong channels. Accordingly, it is an object of this invention to provide a flexible multiplex system which can accommodate signaling lines dedicated to different codes and signaling speeds. Patented Sept. 9, 1969 2 It is another object of this invention to communicate line conditions in the absence of code signaling by way of a multiplex system. It is a further object of this invention to recover syn- chronization without distributing intelligence data to the 5 wrong output ports. In accordance with a feature of this invention, each input port applies a full code character to the bus when scanned. The system thus transmits a character at a time 10 from each input port during each scan cycle. It is a feature of this invention that each input port, after sending a full character to the common bus, staxts the next subsequent input port. The input port determines when a full charcter is transmitted by inserting an ad- 15 ditional final bit in the code character and detecting when this final bit is ready to be applied to the bus. It is another feature of this invention that each output port reads a full character from the common bus and then enables the next subsequent output port. A counter 20 counts each bit as it is read and registered by the output port until the count corresponds to the number of bits in the characters of the code dedicated to the port, whereupon the next output port is enabled. It is a further feature of this invention that an addi- 25 tional.flag bit is inserted by the input port. When a code character is received, the flag bit advantageously corresponds to a parity element in the code. In the

event, however, that a prolonged line condition, such as an "idle" or "break7' condition, is being received, the Rag bit is 30 modified or inverted. This distinguishes the bit sequence for this prolonged condition from code characters, such as "blank" and "rub out," having all elements corresponding to one line condition. The output port, upon registering the flag bit, applies the corresponding prolonged 35 condition to the outgoing line. It is a further feature of this invention that the output port counters and registers are disabled when synchronism is lost to block the distribution of data to the output ports. While regaining synchronization the counters are 40 reenabled whereby the distribution count is maintained although registration of the data is precluded. The foregoing and other objects and features of this invention will be fully understood from the fohowing description of an illustrative 6mbodiment thereof taken 45 in conjunction with the accompanying drawing wherein: FIG. 1 depicts in block schematic form a character at time multiplex system in accordance with this invention. FIGS. 2 and 3 when arranged as shown in FIG. 7 show the details of circuits and equipment which cooperate to .50 form a typical input data port or buffer in accordance with this invention. FIG. 4 ! discloses in schematic form the details of the common transmission path or bus and the input and output common control equipment in accordance with this 55 invention; and FIGS. 5 and 6 when arranged as shown in FIG. 8 show the details of circuits and equipment which cooperate to form a typical output data port or buffer. 60 General description Referring now to FIG. 1, data input leads 101 through 104 depict four of a plurality of incoming lines for the multiplex system. Data input leads 101 through 104 are in turn connected to input ports or buffers 105 through 65 108. The outgoing lines of the system are shown as data output leads III through 114 which leads are connected to output ports or buffers 115 through 118 respectively. The data output leads of input buffers 105 through 108 70 and the data input leads to output buffers 115 through 118 are interconnected by way of a common transmission path or btis shown as metallic line 120. Although bus

3,466,397 3 120 may be a short metallic line as shown in FIG. 1, it is understood that bus 120 may also comprise a long transmission line which rnay include conventional radio or carrier equipment to accept data signals at the input end and reconstruct the data signals at the output end of bus 120. Common to input buffers 105 through 108 is input coriamon control 124 which, as described hereinafter, controls the readout of the input buffers and the generation of the synchronization or framing signal. Common to output buffers 115 through 118 is output common control 125 which, as described hereinafter, detects the framing signal and upon the detection thereof initiates the distribution of the data bits to the output buffers, The system also includes clock 121 which provides the clock pulses therefor. In the event that bus 120 constitutes a prolonged transmission path, it is understood that clock 121 may comprise a master clock at either the ori.-inating end or the terminating end of bus 120 and a slave clock at the other end maintained in synchronization with the master clock in any manner well known in the art. In any event clock 121 comprises a source of pulses which simultaneously distributes clock pulses to all the input buffers, the output buffers, the input common control and the output coriamon control. Assuming now that rharacter signals are being received on the incoming lines such as da-ta Iiiies 101 through 104, each of input buffers 105 through 108 proceed to receive the data characters and store them. Assuming also that the readout of input buffer 108 has concluded, a signal is provided by way of its terminal STS to input common control 124. Input common control 124 in response thereto and under control of the next clock pulse from clock 121 applies a framing signal to bus 120. At the conclusion thereof input common control 124 applies an enabling signal to terminal STP of the first input buffer, namely input buffer 105. This enables input buffer 105 to accept clock pulses by way of terminal CL-1 which clock pulses are utilized to read out one data character by way of the data output lead of input buffer 105. In addition, input buffer 105 provides an additional flag bit which bit may may advantageously indicate the condition of the parity element of the code character. In the event, however, that a code character has not been received by way of data input lead 101, input buffer 105 is alternately arran.-ed to invert the flag bit indicating the signaling condition of lead 101 such as the "break" or "idle" condition. In any event, character bits desi.-nating the code character or the condition of lead 101 are read out by the clock pulses applied to input buffer 105 until all the bits including the flag bit have been applied to bus

120. Thereupon input buffer 105 signals by way of its terminal STS to terminal STP of input btiffer 106. This initiates the operation of input buffer 106 which reads out its character stored therein in substantially the same manner. Accordin,-Iy, the input buffers are sequentially enabled to read out a character at a time to bus 120. When the readout has been concluded by the last input buffer 108, it signals by its output terminal STS as previously described to enable common control 124. This completes a scan cycle, provides a framiiig signal to bus 120 and initiates a new cycle. It is noted that after all the input buffers have concluded their readout, they apply an enablin.- potential by way of their CK terminals to input common control 124. This indicates to input common control 124 that no intermediate one of the buffers are op-,rating and permits input common control 124 to provide a framing signal to bus 120 and initiate a new cycle at the conclusion of the readout by input buffer 108. Recalling now that each cycle is initiated by a framing signal, this framing signal is detected by outplit common control 125 which scans the bus after the bits are distributed to :the output buffers as described hereinafter. Assuming that a correct framing signal is detected, output common control 125 provide5 4n eliabling signal to ter- 4 minal STP of output buffer 115. This enables output buffer 115 to accept clock pulses from clock 121 by way of terminal CL-1. These clock pulses are utilized by output bliffer 11,5 to r.-ad and register the bits applied to the 5' input thereof by bus 120 and to maintain a count of the applied bits. Accordi@i,@ly, since output buffer 115 is started immediately after the framin@ signal, the character bits applied by inplit buffer 105 are read and registered by output buffer 115 for subsequent application to data out- 10 putlead 111. When the count of the bi,s applied by bus 120 correspo-nds to the number of bits of the characters of the codc dedicated to the first buff-ers plus the fla@ bit, the readir@g and registering of the bits are concluded and 15 output buffer 115 provides an enabling signal by way of t,-rminal STS to terminal STP of buffer 116. Accordingly, buffer 116 starts to count, read and register the bits of the character applied to bus 120 by inptit buffer 106. In a sitnilar manner, each of the output buffers sequentially 20 counts, reads and re,@isters the data bits of the corresponding input buffer until output buffer 118 concludes its counting and registration. Thereupon output buffer 118 applies a si.-nal by way of its terminal STS to ouput common control 1-25. This p,-rmits output common con- 25 trol 125 to again read bus 120 to detect the framing si.-nal. Accordingly, at the conclusion of the distributing cycle, output common control 125 examines the framing signal and assuming the framin.- signal is correct, initiates a new distributing cycle. 30 Assuming now that an input buffer has not received a code character the buffer had applied an inverted flaa bit to bus 120 corlesponding to the prolonged condition of the incoming line. Since the corresponding output buffer is concurrently receiving -the data bits, in the event that a 35 code character is not received therein the output buffer examines the flag bit and applies the corresponding prolonged condition to i:ts outgoing line. It is noted that the coi-inting circuit in the output buffer maintains the appropriae count of the number of incomin.@ bits. Accordingly, 40 the distribution cycle is maintained and the next output buffer is enabled to read the appropriate succeeding character. Assuniing now after a cyclc of bit distribution to the output buffers, output comiiion control 125 examines the 45 n--xt bit on bus 120 and finds that it is an incorrect signal indicatin.- loss of synchronization, output common control 125 applies a disabling poteritial to all of terminals DD in output buffers 115 through 118. This has the effect of preeltiding registration of any of the data bits. In addi- 50 tion, output common control 125 passes a disabling potential to terminal DIS of the first output buffer 115. This disables the countin@ circuit in output buffer 115, preeluding -the counting of the bits on bus 120. Output common control 125 then pi-occeds to examine the next suc- 55 cessive bit applied to bus 120 and each successive bit thereaf ter until a correct framing signal is detected. Thereupon output common control 125 removes the disabling potential from terrninal DIS of output buffer 115. Accordingly, outptit buffer 115 proceeds to count the bits 60 applied to bus 120. Re.-istration of the bits is precluded, however, since a disabling potential is still applied to terminal DD. After the appropriate number of bits are counted by output buffer 115, output buffer 116 is signaled, as previ- 65 ously described, to proceed to count the next sequence of bits. Accordingly, the output buffers provide a count of th-@ bit distribution CYCIC WithDut registering any of the bits applied to bus 120. It is noted that during this cycle each of the out,@oing lines are maintained in the signal 70 condition corresponding to the

convention of the output line when synchronization was lost. At the conclusion of the bit distribution cycle, output buffer 118 a.-ain signals OutPut common control 125 and output common control 125 again examines the bit ap- 75 plied to bus 120. Assumminl-, the second bit is a correct

5 framing bit, another distribution counting cycle is initiated although bit registration is still precluded. When this cycle is completed, output common control 125 is again enabled and in the event that the third bit is a correct framing bit, it is presumed that the system is again back in synchronization and the disabling potentials applied to the DD terminals of the output buffers are removed and normal operation is resumed. Input data buffers A typical input data buffer is generally indicated by block 201 in FIGS. 2 and 3. Extending to input data buffer 201 is data input line 202. As previously described, data line 202 is dedicated to a predetermined data code which, it is assumed, is a start-stop code containing a parity bit, in this case for providing even parity. Input data lead 202 extends to the CLEAR input of SM flip-flop 241 which, as described hereinafter, is normally in a CLEAR condition. In addition, input data lead 202 is connected to the first stage of the input data register generally indicated by the blork 208 and to an input of oscillator control circuit 203. ioscillator control circuit 203 is a bistable device having one input thereof extending to data input lead 202 and another input thereof extending to lead 209. The output of oscillator control circuit 203 is connected to oscillator 204. When a negative transition such as a spacing start signal is received over input lead 202, oscillator control 203, in response thereto, is operated to one of its bistable states. In this state oscillator control 203 provides an enabling potential to oscillator control 204, and the action of oscillator 204 is initiated to provide at the output thereof pulses at the bit rate of the incoming signals on data input lead 202. These bit pulses are utilized as shift pulses for input register 208. Oscillator control 203 remains in this state until a negative transition is received over input lead 209 restoring oscillator control 203 to its initial state, and in turn disabling oscillator 204 to terminate the application of shift pulses to input register 208. As previously described, data input lead 202 extends to input register 208. Input register 208 has a plurality of stages numbered in FIG. 2 in accordance with the elements of the start-stop code dedicated for input line 202. As described hereinafter, all of the stages are normally in the CLEAR condition. Viewed from right to left in FIG. 1, the first stage of input register 208 is identified as stage STP to correspond to the start bit of the start-stop code. The succeeding stages are identified as stages 1 through N, and are equal in number to the intelligence elements of the start-stop code. Stag'e P in input register 208 follows stage N and corresponds to the parity bit in the start-stop code and stage SP corresponds to a stop element although the start-stop code dedicated to input lead 202 may contain more than one stop element. With data input lead 202 extended to input register 208 and more particularly to stage SP by way of lead 206, a combination of the application of a marking condition by data input lead 202 to stage SP and the transition of the output of oscillator 204 from a low condition to a high condition clears stage SP. Conversely, when an input spacing condition is applied to stage SP together with a shift pulse transition, stages SP is SET. Accordingly, stage SP stores a spacing bit therein when it is SET and stores a marking bit therein when it is CLEARED. Similarly, all other stages of input reigster 208 store spacin-, bits in the SET condition and marking bits in the CLEAR condition. Assuming now that a start-stop character is received from data input line 202, when the start bit is received the consequent negative transition of the line drives oscillator control 203 to the first bistable state as previously described, thereby enabling oscillator 204. Oscillator 204 in ttirn starts to operate providing a shift pulse at the 3,466,397 6 theoretical midpoint of the start element and, since it is operated at the bit rate of the incoming channel, at the theoretical midpoint of each succeeding element. Accordingly, at the theoretical midpoint of the start element, stage SP of input reigster 208 is SET. When the first intelligence element is received and at the theoretical midpoint thereof, oscillator 204 produces the next shift pulse driving stage SP into the condition corresponding to the first element and entering the start pulse in stage P by 10 setting the stage. Similarly, each of the succeeding intelligence elements, the parity element and the stop bit is entered into stage SP of input register 208, and each prior element is shifted down through the register until the start bit is stored in stage STP, the intelligence bits 15 are stored in

stages 1 through N, the parity bit is stored in P and the first stop bit is stored in stage SP. The entering of the start bit into stage STP transfers the condition of the stage from the CLEAR to a SET condition. This drives the "O" or CLEAR output ter- 20 minal of stage STP from the high voltage to the low voltage condition. The consequent negative transition at the "O" output of stage STP is passed to lead 209 and thus to oscillator control 203. Accordingly, as previously described, oscillator control 203 is restored to the initial 25 bistable state disabling oscillator 204 and thus terminating the application of shift pulses to input register 208. Thus, the entering of data bits into input register 208 is terminated until the next negative or spacing condition- transition on data input line 202. 30 When the start pulse is entered into stage STP, driving the stage from a CLEAR to the SET condition, the SET or "I" output terminal of stage STP goes from a low voltage to a high voltage condition. This high condition is provided to delay circuit 211 by way of lead 210, 35 and after a predetermined delay, is then provided to one input terminal of AND gate 212. The other input terminals of gate 212 extend to leads 214 and 215. Lead 214, is the "not" clock lead which extends to the output of clock 401, FIG. 4. Clock 401 provides at its 40 output clock lead conventional clock pulses and its output "not" clock lead inverted clock pulses, that is, the pulses on the "not" clock lead correspond to the interpulse periods on the clock lead. The pulse repetition rate of clock 401 determines the bit rate of the common bus 45 and is therefore slightly in excess of the cumulative signaling rate required to acommodate the signals received on all the input channels. As described above, lead 214 applies a positive condition to gate 212 during the interpulse clock period, thus 50 enabling gate 212 during this interpulse period. This is arranged to provide that the subsequent operation of input register 208 and the readout thereof does not occur during other operations of the input data buffer 201 which operations are initiated by the clock pulses. '55 Returning now to gate 212, input lead 215 extends to the "O" or CLEAR output of RM flip-flop 321. As de. scribed hereinafter RM ffop-flop 321 is in the CLEAR condition when data is not being read out onto the common bus. Assurning therefore that data is not being read 60 out, RM flip-hop 321 is in the CLEAR condition, lead 215 is in the high condition and gate 212 is enabled. Accordingly, the output of gate 212 is driven to the high condition in response to the delayed- transition from stage STP. This condition is passed to the CLEAR input of 65 stage STP restoring it to the CLEAR condition. The restoration of stage STP to the CLEAR condition drives the "O" output thereof to the high condition. This positive transition is applied to monopulser 218 and monopulser 218, in turn, generates a positive pulse at 70 the output thereof. This positive pulse is passed by way of lead 219 to the CLEAR inputs of stages I through N, P and SP in input register 208. Accordingly, all of the stages of input register 208 are restored to the CLEAR 7.5 coridition in preparation for the next reception of signals from data inptit lead 202.

3,466,397 7 The output pulse provided by monoplilser 218 signals the completion of the storage of the inptit start-stop character in input register 208 and provides the readout or gating pulse. This gating pulse is passed from the output of monopulser 218 to lead 220 and thence to a gate generally indicated by block 301, FIG. 3. In general, gate 301 functions to read out the character from input register 208 into the bus register generally indicated by block 320. Bus register 320 is viewed frorn right to left in FIG. 3 includes stages I throu.-h N correspondin.- to stages I through N in input register 208 and sta.-e F. Stages I through N correspond to the data bits in the start-stop code and stage F corresponds to a flag bit added to the code character as describe-d hereinafter. The flag bit entered at stage F is dependelit upon several condil@ions such as the parity bit, the condition of data input line 202 and certain of the codes received therefrom. Returnin- now to -ate 301, it is noted that the gate includes AND gates 311 through 314 and 315 throu,@h 318. Considering first gates 311 through 313, one inplit of each of these gates extends to lead 220 which, as previously described, provides the gating piilse. The other input leads to gates 311 through 313 extend by way of leads 221 throu,@h 223 to the "I" outputs of stages 1 through N. The outputs of gates 311 through 313 then extend by way of OR gates 302 to 304 to the SET inputs of stages I through N of bus register 320. Accordingly, gates 311 through 313 and the intermediate gates therein, not shown, function in response to the gite pulse on lead 220 to set stages I through N of bus register 320 in the event that corresponding stages of I through N of input registers 208 are SET. Thus, a spacin.- bit stored in a stage in input register 208

is read out and stored in a correspondin.- stage in bus register 320. Gate 301 also includes AND gates 315 through 317, and these gates similarly have one input thereof connected to lead 220. The other inputs to gates 315 through 317 extend by way of leads 231 through 233 to the "O" outputs of stages I through N of input re.- ister 208. Since the outputs of gates 315 through 317 pass through OR gates 306 through 308 to the CLEAR inputs of sta.-es I tbrough N of bus register 320, ,ates 315 through 317 function to pass marking bits in stages I through N of input register 208 to corresponding sta,-es iii bus regis 1 er 320. As described hereinafter, gates 314 and 319 operate to insert the appropriate flag bit in stage F of bus register 320. Summarizing, therefore, it is seen that after th-, start-stop code character is entered in input register 208, and when bus re-ister 320 is not being read out, input register 208 is read out through gate 301 into bus-register 320 and register 208 is restored to the CLEAR condition to await the next signal from data indut line 202. The readout of bus register 320 onto the comn-ion bus occurs after the input data buffer prior to buffer 201 completes its readout or, in the event that input data buffer 201 is the first buffer, then after the common control applies its framing signal to the common bus. Upon the completion of the readotit of the prior buffer, or where data buffer 201 is the first ibuffer, then upon the application of the framing signal to the bus a positive pulse is received on terminal STP and thus applied to lead 322. The positive pulse on lead 322 is extended to the SET input of RM flip-flop 321, placing the flip-flop in the SET condition, it being recalled that the flip-flop is in the CLEAR condition prior to readout. In addition, the pulse on lead 322 is passed by way of lead 324 to the CLEAR input of M flip-flop 323. M flip-flop 323, which is normally in the SET condition as described hereinafter, is thus placed in the CLEAR condition. It is no@ed at this time that M flip-flop 323 has an output extending to bus register 320 and more particularly to stage F. It is arranged that when shift pulses are applied to stage F, the stage is driven to a condition in accordance with the condition of M flip-flop 323. 8 Recalling now that the start of readout pulse on lead 322 sets RM flip-flop 321, the "O" output thereof is driven to a low voltage condition, which condition is ap@ plied to otitptit terininal CK and to lead 215. As previous- ly described, the low voltage condition on lead 215 disables gate 212 to preclude the readout of the input register. The function of output terniinal CK will be described hereinafter. With RM flip-flop 321 in the SET condition, the "I" out- 10 put terminal thereof is driven to the high condition, which condition is applied to AND gate 326. Gate 326 comprises the readout gate and with RM flip-flop 321 SET, readout gate 326 is erabled to read out the conditions of the first stage in bus register 320. Accordingly, the output of gate 15 326 will go to the high coiidition when the first sta,@C is CLEAR and to the low condition when the first sta,@e is SET. Thus the storage of a mark bit in the first stage of bus regist@r 320 applies a positive condition to lead 328 and then to terminal BI which, as described here- 20 iriafter, is connected to the common @bus. The "O" outpi-it terminal of RM flip-flop 321 is also connected to an input of OR gate 325. Recalling now that RM flip-flop 321 is normally in the CLEAR condition and the "O" output thereof is in the high voltage 25 condition, this high voltage condition is thus passed through OR gate 325 to lead 340. The other input to gate 325 is connected to the clock output by way of lead 327. With RM flip-flop 321 in the CLEAR condition, however, output lead 340 of lead 325 is n-iaintained in the high con- 30 dition whereby the application of the clock pulses thereto is precluded. When RM flip-flop 321 is SET and the "O" output thereof is driven to the low voltage condition, the high voltage condition thus applied to lead 340 is removed. 35 Accordingly, clock pulses applied by way of lead 327 are now passed by gate 325 to lead 340. It is noted here, as described heteinafter, that the high condition applied by RM flip-flop 321 is removed simultaneously with the application of the leading edge of the 40 clock pulse to lead 327. Accordingly, the first low voltage to high voltage transition on the lead 340 does not occur until the initiation of the next subsequent clock pulse. RM flip-flop 321, however, has been SET, and consequently gate 326 has been enabled for a full bit period 45 prior to this transition. Accordin.-ly, the condition of the first stage of bus register 320 is read out before the transition occurs on lead 340. This first bit is applied by way of lead 328 and terminal BI to the common,bus. Lead 340 extends to the shift pulse input of bus regis- 50 ter 320 and to the SET input of M flip-flop 323. The above-described next subsequent shift pulse, that is, the first low to high transition on lead 340 thus sets M flipflop 323 and provides the first shift pulse for bus register 320. This shift pulse therefore inserts the marking bit

5@3 from M flip-flop 323 into stage F, shifts the flag bit from stage F to stage N and shifts the conditions from each stage to each prior stage whereby the condition in stage 2 is shifted to stage 1. The second bit is thus read out of btis register 320 through gate 326 to the common bus. 60 Upon the application of the next subsequent shift pulse to bus register 320, each bit is similarly shifted forward. Since M flip-flop 323 is now in the SET condition, however, a spacing bit is read into stage F. Simultaneously, th@- marking ibit initially read into stage F is shifted into 65 stage N. For each succeeding shift pulse, the marking bit initially stored in M flip-flop 323 and read into stage F is passed from stage N to succeeding stages. In addition, with M flip-flop 323 SET, spacing bits are read into stage 70 F and shifted down through the stages to follow the marking bit. Accordingly, as the code character is shifted down through bUS Tegister 320 followed by the flag bit, a marking bit is shifted down immediately following the fla.- bit, and the stages subsequent thereto fill up wi'h 7 -) spacing bits. Thus, the code character continues to be

9 3,466,397 10 shifted down in bus register 320 until the flag bit enters stage 1, the marking bit enters stage 2 and the stages subsequent thereto are filled with spacing bits. At the completion of the readout of the flag bit by gate 326, the next shift pulse transition is applied to lead 340, moving the marking bit to stage I and filling all subsequent stages with spacing bits. The "1" output termitials of all subsequent stages together with "I" output of M flip-flop 323 are therefore in the high voltage condition. These, terminals are all connected to AND gate 345. The output of AND gate 345, therefore, goes to the high voltage condition, which condition is applied to the CLEAR input of RM flil3- flop 321. RM flip-flop 321 is therefore CLEARED, disabling gate 326, reapplying the high voltage potential through OR gate 325, reenabiing gate 212 and reapplying the high voltage condition to terminal CK. It is noted that this clearing of RM flipflop 321 occurs simiiltancously with the application of the leadin.- edge of the shift pulse since this shift pulse shifted the marking bit out of stage 2 to enable gate 345 to clear RM flip-flop 321. Thus, lead 340 which was in the hi-h voltage condition in response to the shift pulse is maintained in the high voltage condition by RM flipflop 321. The output terminal of RM flip-flop 321 is also connected to monopulser 346. When RM flip-flop 321 is CLEARED, the positive voltage transition at the "O" output thereof is applied to monopulser 346 which generates at its output thereof a positive p,,ilse. This positive pulse is applied through lead 347 to output terminal STS. As previously described, terminal STS of each input data buffer is connected to terminal STP of each subs--quent input buffer with the exception of the last buffer wherein terminal STS is connected to common control. e' Accordin ly, upon the termination of r adout and the restorin.- of RM flip-flop 321 to the CLEAR condition, monopulser 346 sends a positive pulse to the STP terminal of the next subsequent buffer or to common control. This initiates the readout of the next subsequent buffer in the same manner as previously described with respect to input data buffer 201. The positive pulse at the output of monopulser 346 is also passed by way of lead 348 to gates 242 and 244, FIG. 2. As previously described, SM flip-flop 241 is normally in the CLEAR condition. With the "O" output terminal thereof connected to @ate 242, this gate is enabled. Conversely, with the "1" output terminal thereof connected to gate 244, this latter - ate is disabled. Accordingly, in the normal condition, the pulse on lead 348 is pass- ,d through gate 242 to lead 243. Lead 243, in turn, is connected to OR gate 305 and to OR gates 306 through 308. Since the output of OR gate 305 is connected to the SET input of stage F and the outputs of OR gates 306 to 308 are connected to the CLEAR inputs of stages I to N in bus register 320, sta,@e F is placed in the SET condition and stages I to N are restored to the CLEAR condition. Accordingly, at the termination of readout, stage F is rormally in the SET condition and stages I-N of bus register 320 are normally res@ored to the CLEAR condition in preparation for the next readin.- of the character in input register 208. In addition, with RM flip-flop 321 in the CLEAR condition, it is ready to respond to another pulse from terminal STP to again read out the charaCteT stored in'bus register 320 to the common bus. Summarizing the above-described op,-rations, the information elements of the code character received and stored by input register 208 are Tead out and transferred to bus re-ister 320. In the process the start and stop ele,ments are stripped off, the parity element is examined as described hereinafter, and a new flag bit is inserted in bus register 320. Thereafter, in response to a signal from the prior input data

buffer or from common control if buffer 201 is the first channel, the intelligence elements and the flag bit are read out o-@ito the bus, and, at the conclusion thereof, the subsequent input data buffer is signaled to initiate its readout. Accordingly, each input buffer is assi-ned a plutality of sequential time slots, the number of time slots corresponding to the number of intelligence eiements for the code character dedicated to the input lead for the bliffer plus one flag bit. Thus, for each read- out cycle the input buffers sequentially read out a char- acter at a time to the bus. Flag bit insertion 10 As previously indicated, the flag bit entered in bus re@ister 320 depends on the input code characters, the condition of the input line and/or the parity bit received by input register 208. In the event that the input line is in the idle marking co.,idition, a "I" or spacing bit will 15 invariably be inserted in stage F of bus register 320. This is to insure that the continuing condition wherein "O" or. marking bits are contained in stages I through N, a spacidg or "I" bit will be continuously inserted in stage 20 F to indicate the idle line condition. In the event, however, that a "rub out" o-r "lettcrs" character is received, which character contains all markina intelligence elements, then a "O" or marking bit will be ins-,rted in stage F. Thus, a "rub otit" will be clearly distinguishable from an idle line@ condition. 25 When the incoming line is in a prolonged "break" or spacing c ondition, then a "W' bit is inserted in stage F. Thus, during the "break" condition, "I" or spacing bits are read into stages 1 through N while a "O" or markinbit is inserted in stage F. When a "blank" character is 30 received wherein all intelligence elements are spacing elements, a "1" or spacing bit is inserted in stage F. This prmits the "blank" character to be distinguished from the prolonged "break" or spacing condition. During normal signaling sequences, a "1" bit is inserted 35 in sta-e F when the imput start-stop code parity bit is "O" or marking, and a "O" bit is inserted in sta,-e F when the parity bit is "I" or spacing. This normal signalin-. condition involves all situations excludin."idle", "break," "letters" character or "blank" character 40... receiving situations. Assuming now that input line 202 is idle, oscillator control 203 does not enable oscillator 204. Accordingly, no spacing bit is inserted in input register 208, and monc)pulser 218 therefore does not provide any gating pulse 45 to lead 220. It is recalled that, upon the completion of readout, monopulser 346 applies a pulse to lead 348 which pulse passes through gate 242 to lead 243. This pulse is then applied to gates 305 through 308 inserting 50 marking bits in stages I through N and a spacing bit in stage F. Thus, with input lead 202 in the idle condition precluding the application of a gatin-. pulse to lead 220, stages I through N are maintained in the CLEAR condition, and stage F is maintained in the SET condition. 55 Accordingly, upon the next readout of bus register 320, marking bits corresponding to stages 1 through N will be read out followed by the spacing flag bit stored in stage F. At the completion of readout, monopulser 346 w . ill a.-ain pass a pulse through gate 242 and marking Go bits will again be inserted in stages I through N and a spacing bit will be inserted in stage F. . Assu . ming now that a "rub out" or "letters" character is received, the start element of the character operates oscillator control 203 to enable oscillator 204. Accord- C5 ingly, after the character is completely read into input register 208 and the start element is inserted in stage STP, monopulser 218 is opera@ed, as previously described, to apply a gate pulse to gate 301. The intelligence ele- ments of the character are thus read from input register 70 208 into bus register 320. Since all the intelligence elements of the "letters" character are marking, the "O" outputs of stages 1 through N in input register 208 are in the high condition. These outputs are all connected to gate 251 whereby the output thereof goes to the high 75 condition which condition is passed through OR gate

3,466,397 252 to lead 253. Lead 253 in turn extends to an input of AND gate 318 in gate 301. Since the other input to AND gate 318 is connected to lead 220, the high condition on lead 253 enables -ate 318 to pass the gate pulse therethrough and through OR gate 309 to the CLEAR input of stage F of bus register 320. Accordingly, when a "rub out" or "letters" character is received, the marking elements are inserted in bus re-,ister 320, and a marking fla.- bit is also inserted in stage F. When a prolon.-ed "break" or spacing condition is received, the initial mark to space transition operates oscillator control 203 and oscillator control.203 in turn enables oscillator 204 to apply shift pulses to input register 208. This results in the insertion of spacin.- bits in input register 208 since input line 202 is maintained in the spacing condition. Accordingly, at the conclusion of one character interval, a

simulated spacing start bit is fed into stage STP and monopulser 218 thus applies a gati-Tig pulse to lead 220. Gate 301 therefore reads out the spacin.@ bits in input re.cister 208 and inserts them in bus re.-ister 320. With the "break" condition in inptit lead 202, the simulated character inserted in input register 208 does not include a stop element. Accordingly, a spacing bit is inserted in stage SP of input register 208. This drives the "1" output thereof to the high condition, which condition is passed through OR gate 252 to lead 253. Accordingly, gate 318 is enabled upon the application of the gating pulse to insert a markin.- bit in stage F. Thus, in response to the initiation of the "break" signal, spacing bits are inserted in stages I throu@h N of bus re.-ister 320 and a markin- bit is inserted in sta.-e F. Since the "break" condition is reco.-nized by input re_gister 208 as a stream of spacing bits, the "I" outputs of stages I thro3i.-h N are in the hi,-h condition. These "I" outputs are all connected to gate 255 providing a high condition at the output thereof. This high condition is fed to one input of AND gate 258. The other two inpi-its to AND gate 258 are connected to the "I" outputs of stages P and SP in input register 208. Since only spacing bits have been fed to input register 208, these I'll, otitputs are also in the hi,-h condition. Accordingly, the output of gate 258 also goes to the high condition, which condition is fed to one input of AND .@ate 259. Since the other input to AND gate 259 is connected by way of lead 230 to lead 220, the -ating pulse produced by monopulser 208 is applied by gate 259 to the SET input of SN flip-flop 241. Accordingly, the reception of the initial transition of the "break" si.-nal affects the setting of SN flip-flop 241. With this flip-flop SET, the "O" olitput goes to the low condition, disabling gate 242, and its "I" output goes to the high condition, enabling -ate 244. It is recalled that a "break" signal constitutes a prolonged spacing condition. Accordingly, when the previously described character interval is terminated, the stages of input register 208 are CLEARED and oscillator control 208 is restored to its initial condition as previously described. Since input lead 202 stays in the spacing cond . ition, there is no subsequent mark to space transition to enable oscillator control 203. Accordingly, oscillator 204 is not reenabled to apply more shift pulses to input register 208. Thus, after the first character interval, subsequent spacing bits are not inserted in input register 208 and monopulser 218 is not operated to generate subsequent gating pulses. Recalling now that at the conclusion of readout from bus register 320 monopulser 346 is operated, the output pulse thereof applied to 1--ad 348 cannot pass through gate 242 since this gate is disabled by the clearing of SN flip-flop 241. With gate 244 enabled, however, the ptilse on lead 348 is applied therethrough to lead 245. Lead 245 in turn is connected to OR gate 309 whereby the pulse applied thereto clears stage F. In addition, lead 245 is connected to OR gates 302 through 304, setting stages I throu.-h N in response to the pulse on lead 245. Thus, 12 although after the readout of the "break" signal subsequent gating pulses are not applied to gate 301, the settin@ of SN flip-flop 241 and the consequent enabling of gate 244 functions to insert spacing bits in stages I through N of bus register 320 and insert a marking bit in stage F. Accordin.-ly, tipon each subsequent readout, the character inserted in bus register 320 corresponds to the "break" condition. At the concliision of the "break" condition, input lead 202 restores to the marking condition. This space to 10 mark transition is applied to the CLEAR input of SN flip-flop 241. SN flip-flop 241 is thus restored to the CLEAR condition disabling AND gate 244 and reenabling AND gate 242. The circuit is thus restored to the initial 15 condition prior to the recept,on of the "break" signal. Assuniin- now that a "blank" character is received, the- recep'lion of tiie start element of the "blank" character enables oscillator control 203 -which iii turn enables oscillator 204. Accordingly, the "blank" character is read into 20 input register 208, and the insertion of the start element in sta,@e STP operates monopulser 218. The operation of monopulser 218 provides a gate pulse to gate 301, and ,,ate 301 reads all the spacing bits out of input register 208 aid into bus register 320. The "blaiik" character includes a stop pulse whereby 25 stage SP provides a low condition to AND gate 258 thereby disabling the gate and provides a low condition to OR .-ate 252. Since all the intelligence elements are spacin,-, gate 255 provides a bigh condition at the output thereof. The high condition at the output of gate 255 is 30 ' applied to inverter 257 which in turn passes a low condition to AND gate 256 disabling this gate. Accordingly, gate 256 applies a low condition to OR gate 252. Since the intelligence elements are not marking, gate 251 provides a low condition at the output thereof as previously 35 described, and this low condition is applied to OR gate 252. ThLis, all of the inputs

to gate 252 are in the low condition, and this low condition is passed to lead 253. Lead 253 in turn is connected to the input of inverter 40 350, and inverter 350 thus applies a h-gh condition to the input of gate 314. Accordingly, gate @114 is enabled, and with the other input th@,reof connected to lead 220, gate 314 passes the gate pulse therethrough and through OR gate 305 to the SET input of stage F of bus register 320. 45 Accordingly, in response to the reception of a start-stop "blank" character, spacing bits are inserted in stages I through N of bus register 320, and a spacing flag bit is inserted in stage F. AssumiDg that a start-stop code character is received other than "blank" or "letters," neither gates 251 nor 50 255 are enabled as previously described. With the output of gate 255 in a low condition, however, inverter 257 applies a high condition to AND gate 256 thereby enablidg the gate. If the start-stop character received contains a marking parity bit, sta.-e P of input register 208 is CLEAR. Accordingly, a low condition at the one input thereof is applied to AND gate 256, and AND gate 256 applies a low condition to OR gate 252. Since the other inputs to OR gates 252 are also in the low condition, as previously 60 described, lead 253 goes to the low condition and inverter 350 enables AND gate 314 as previously described. Thus, the -ate pulse is passed by gate 314 through gate 305 to set stage F. Accordingly, when a marking parity bit is 65 received by input register 208, a spacing flag bit is inserted in stage F of bus register 320. When a spacing parity bit is received by input register 208, the "1" output of stage P goes high, enabling AND gate 256 since inverter 257 is applying a high condition to o the other input of AND gate 256. As i)reviously described, the high condition at the output of gate 256 is passed by way of OR gate 252 and lead 253 to gate 318. Gate 318 therefore is enabled to pass the gating pulse through OR gate 309 to the CLEAR inptit of stage F of bus register 75 320. Thus, when a sp@icing parity bit is received by input

3,466,397 13 register 208, a marking fla- bit is inserted in stage F of bus register 320. Input common control Referring now to FIG. 4, lead 403 constitutes the com mon bus. Bus 403 is connected to the output of OR gate 402. The inputs to OR gate 402 are connected to the BI terminals of the input data buffers and to input common control 406. Accordingly, OR gate 402 passes the data readout from the input data buffers to bus 403, and, in addition, passes a framing pulse from input common control 406 to bus 403 as described hereinafter. As previously described, the input common control sbown generally as block 406 in FIG. 4, inserts the framing bit at the conclusion of the readout of all the input data buffers. This framing bit is alternately marking and spacing. Thus, at the conclusion of any readout cycle, if the framing bit is a marking pulse, the framing bit at the conclusion of the next readout cycle is a spacing pulse, and then at the conclusion of the next readout cycle, another marking pulse. It is recalled at the conclusion of the readout of the last buffer the monopulser corresponding to monopulser 346 applies a pulse to its output terminal STS-N. It is also recalled that as each input buffer concludes its readout the RM flip-flop corresponding to flip-flop 321 is cleared, driving the CK terminal to the high condition. Accordingly, after the readout of the input buffers all of the CK terminals are in the high condition, and a pulse is applied to the STS terminal of the last input data buffer. Referring now to FIG. 4, it is seen that the leads extending to the CK terminals are connected to AND gate 414 in input common control 406. In addition, the lead extending to terminal STS-N is connected to AND gate 415. With all the CK leads in a high condition, the output of gate 414 goes high, enabling AND gate 415. Accordingly, the pulse from terminal STS-N passes through AND -ate 415 to the CLEAR input of SW flip-flop 408. With SW flip-ffop 408 driven to the CLEAR condition, the "1" output terminal goes to the low condition, removing the high condition previously applied to OR gate 411. Concurrently, the "O" output of SW flip-flop 408 goes to the high condition, and this transition is applied to the toggle input of SB flip-flop 410. The high condition at the "O" output of SW flip-flop 408 is also extended to one input of AND gate 409. The application of the positive transition to the toggle input of SB flip-flop 410 transfers the state of the flip-flop. Thus, if the flip-flop was in the CLEARED condition, it is now driven to the SET condition, and, conversely, it is driven to the CLEAR condition ff it previously was in the SET condition. Assuming that SB flip-flop 410 is driven to the CLEAR condition, the resultant high condition at its "O" oiitput is applied to AND gate 409. Accordingly, the output of AND gate 409 goes to the high condition, which condition is applied to OR gate 402. The application of the high condition to gate

402 is passed therethrough whereby a marking framing bit is applied to bus 403. Conversely, if SB flip-flop 410 is driven to the SET condition, the "O" output thereof is in the low condition, and the output of gate 409 is low. Accordingly, a low con-dition is applied to gate 402, passing a spacing framing bit to bus 403. Upon the initiation of the next clock pulse by clock 401, a positive transition is applied by way of the clock lead, to OR gate 411. Since SW flip-flop 408 has been cleared and the positive condition applied by way of its "I" output to OR gate 411 has been removed, a positive clock pulse transition is passed to the SET input lead of SW flip-flop 408. The consequent setting of the flip-flop drives its "I" output back to its high condition and monopulser 416 responds by providing a positive pulse to output lead STP-1. This lead, as previously described, extends to the first input data buffer, and functions to set 14 the RM flip-flop therein to initiate its readout cycle. With SW flip-flop 408 now SET, its "O" output goes to the low condition disabling gate 409. This terminates the framing pulse provided by the input common control 406. 5 At the conclusion of the readout cycle of the input buffers, all the CK terminals are again in the high condition and the last input buffer applies a pulse to the STS-N terminal. Accordingly, SW flip-flop 408 is again CLEARED, the state of SB flip-flop 410 is reversed and 10 another framing bit, having a condition opposite the prior framing bit, is applied to bus 403. Thereafter, on the next clock pulse, the framing bit is terminated and the first input buffer is started to initiate the next readout cycle. 15 Output common control The signals supplied to bus 403 are distributed to the several output data buffers and the output common control generally indicated by block 418. Output common 20 control 418 functi-ons to determir@e whether the framing bit comprises alternate marking and spacing bits and, if the framin.- bits are correct, initiate the operation of the first output data buffer to read the first character in the scanning cycle. In the event that the framing bits are 25 not alternated, output common control 418 is arranged to scan successive bits until the proper alternating sequence is detected. This "slipping" of time slots functions to restore proper framing. The operation of output common control 418 is started 30 when the last output data buffer receives its data charac-ter. The last output data buffer thereupon sends a signal to output common control 418 by way of terminal STS-N, as described hereinafter. This signal is applied by way of lead 419 to set ECC flip-flop 420. In addition, since 35 lead 419 extends to the toggle input of ST flip-flop 421, this latter flip-flop is reversed in its condition. ST flil),- flop 421 remembers the alternate states of the framing bit. With ST flip-flop 421 in its SET condition, a marking framing bit is anticipated whereas when ST flip-flop 421 40 is in its CLEAR condition, a spacing framing bit is ex- pected. In accordance therewith, with ST flip-flop 421 SET and a marking framing bit received by way of bus 403, the high condition of output terminal "I" of ST flip-ffop 421 enables gate 426 to pass the marking framing 45 bit therethrough to OR gate 428. OR gate 428 in tum applies an enabling potential to the SET input of stage A of sbift register 430. Although stage A is not set until a shift pulse is applied thereto, the enabling of the SET input indicates that the proper framing bit has been 5o received. Conversely, with ST flip-flop 421 CLEARED, and a spacing framing bit received from bus 403, output terminal "O" of flip-flop 421 enables gate 427. Inverter 425 inverts the spacing framing bit, passing it throliah enabled gate 427 and OR gate 428 to enable the SET 55 input of stage A of shift register 430. Thus, it is indicated that. the correct framing bit is received. Returning now to the setting of ECC flip-flop 420, the conseqlient high condition at output terminal "I" thereof enables gate 442. Thereafter, the "not" clock 60 impulse is passed through gate 442 and OR gate 443 to provide a sbift pulse to shift register 430. Accordingly, the previously described enabling of the SET input of stage A functions with the shift pulse to set stage A. Upon the next occurrence of the clock impulse, OR 65 gate 422 ai)plies an enabling pulse to the CLEAR input of ECC ffip-ffop 420. The "O" output thereof is thus driven to the high condition, and monopulser 455 in response thereto applies a puls-. to output terminal STP-1. This signals the termination of the framing pulse, and 70 the pulse on terminal STP-1 initiates the operation of the first output data buffer to read the first data character in the cycle. With ECC flip-flop 420 in the CLEARED condition, a high condition is also applied to OR gate 422. This renders the output of OR gate 422 unrespon- 75 sive to the clock pulses. In addition, with ECC flip-flop

15 420 CLEARED, AND gate 442 is disabled to block the ".not" clock impulses to shift

register 430. In the event that synchronism is lost and incorrect framing pulses are received, output common control 419 examines the subsequent time slots until a bit having the correct condition is detected. This action, however, is not initiated until two successive incorrect framing bits are detected. Assuming now that ST flip-flop 421 is SET by a piilse over lead 419 and a spacing framing bit is received, gate 426 is enabled, but a high condition pulse is not applied thereto by bus 403. Accordin,-Iy, the output of OR gate 428 goes to the low condition wbereby inverter 429 provides an enabling signal to the CLEAR input of stage A of shift register 430. Accordingly, wlien the "not" clock impulse is applied throu.-h gates 442 and 443, stage A is CLEARED. Conversely, if ST flip-flop 421 is in the CLEAR condition in anticipation of a spacing framin@ bit and a marking framin@ bit is received, gate 427 is Inabled, as previously described, but inverter 425 applies a low condition thereto. Accordingly, the output of OR -ate 428 is in the low condition, and inverter 429 applies an enable potential to the CLEAR input of stage A. Thus, stage A is CLEARED in the event that an improper framing bit is received. When the next successive framing bit is anticipated, a pulse is again applied to lead 419 to the toggle input of ST flip-flop 421 thereby reversing its condition. If, at this time, the correct pulse is detected, stage A will be SET, and the circuit action will continue in the normal manner. In the event, bowever, that an i-ticorrect framing bit is detected, an enabling potential will be applied by inver-ter 429 to the CLEAR input of stage A in the same manner as previously described. - Accordin,-Iy, upon the reception of the "not" clock impulse, the sbift pulse applied by OR gate 443 places stage A in the CLEAR condition and sbifts the previous CLEAR condition of sta,@e A to sta,@e B. With both stages A and B of register 430 in the CLEAR condition, the "O" output terminals thereof go to the high condition to enable AND gate 450. Gate 450 in turn is connected to one input of each of AND gates 451 and 452. The other input to each of AND gates 451 and 452 are connected to the clock lead. Accordingly, the next subsequent clock imi3ulse enables AND gate 451 to clear DD flip-ffop stage 4S9 and set DS flip-flop stage 434. The clearing of DD flip-flop 439 brings its "I" output terminal to a low condition. This low condition is applied .to terminal DD which terminal extends to all the output data buffers. As described hereinafter, with terminal DD in the low condition, the input gate to the registers in each of the output data buffers is disabled, precluding the re@istration of subsequently received characters. Returning now to the setting of DS flip-flop 434, output terminal "O" is driven to the low condition. This low condition is extended to terminal DIS which terminal extends to the first output data buffer. As described hereinafter, the application of the low condition to terminal DIS disables the first output data buffer whereby the buffer cannot initiate a count of the incoming data bits, and therefore is precluded from providing the stepping of the readout cycle and the subsequent enabling of the second output data buffer. Accordirigly, with DS flip-flop 434 SET, the readout cycling of the output data buffers is halted. The setting of DS flip-flop 434 also drives its "I" output to the high condition to enable gate 444. ThLis, when the "not" clock impulse is received this pulse will be passed by gate 444 to OR gate 443 to provide a shift pulse. T'he clock pulse which set DS flip-flop 434 and cleared DD flil)-flop 439 also cleared ECC flip-flop 420 as previously described. With the first output data buffer disabled, however, the consequent pulse provided by monopulser 455 to enable the first output data buffer does not initiate 3,466,397 16 the readout cycle. The first bit, however, although not registered by the first output data buffer, is examined by gates 426 and 427 and since ST flip-flop 421 remains in its prior condition. In the event that this still is not a proper framing bit, the next "not" clock pulse with gate 444 enabled again clears stage A of shift register 430. Accordingly, output common control 418 maintains the output data buffers disabled and slips another data bit. The next data bit is therefore again examined, and this 10 process is repeated. Assuming now that a data bit corresponding to a proper framing bit is detected, an enabling potential is applied to the SET input of stage A. The "not" clock impulse then applied through gate 444 and OR gate 443 sets 15 stage A, driving the output thereof to the hi,-h condition. This enables gate 433 which, upon the reception of the clock implilse, clears DS flip-flop 434. With DS flip-flop 434 CLEARED, the counting of the first output data buffer is enabled and the output data buffers maintain 20 a count of the readotit cycle although the registration of the characters is precluded since DD flip-flop 439 is maintained in the CLEAR condition. In addition, with DS flip-flop 434 CLEARED, the "I" output thereof goes to the low condition, disabling AND gate 444. The appli- 25

cation of shift pulse to shift register 430 is therefore terminated, thereby concluding the detectin,@ of the data bits received from bus 403. At the conclusion of the readout cycle, the last output data buffer again applies an impulse to lead 419 setting 30 ECC flip-flop 420 and changing the condition of ST flipflop 421. Accordingly, the next framing bit is examined in the same manner as previously described. If the framing bit is incorrect, stage A is again CLEARED, and the previously described process is re- 35 peated wherein DS flip-flop 434 will be SET in the event that two successive incorrect framing bits are detected, and, as a result thereof, output common control 418 slips time slots to detect a correct frarning bit. Assuming, however, that a second consecutive correct 40 framing bit is detected, the "not" clock impulse provides a shift pulse to shift register 430 as previously described whereby stages A and B are SET. The subsequent clock pulse then clears ECC flip-flop 420 thereby starting a new readout cycle. With DD ffip-ffop 439 still cleared, 45 the data characters are still not registered by the output buffers, bowever. At the conclusion of this readout cycle, the last output data buffer again sets ECC flip-flop 420 and switches the state of ST flip-flop 421. Accordingly, ie next fram- 50 ing bit is examined. Assuming that this framing bit is correct, the "not" clock impulse sets stage A, and shifts the previous SET conditions of stages A and B to stages B and C respectively. Accordingly, all of the stages in shift register 430 are SET. This is examined by AND 55 gate 437 whose inputs are connected to the output I'll, terminals of stages A, B and C. Since all these terminals are in the high condition, gate 437 enables gate 438. The clock pulse is concurrently being applied to gate 438. 60 Accordingly, gate 438 sets DD flip-flop 439. This restores the "I" output of DD flip-flop 439 to the high condition and with terminal DD in the high condition, the oiitput data buffers are enabled to register the data characters. Accordingly, when the circuit falls out of 65 synchronism, tbree successive correct framing bits are required to restore the circuit to the normal condition. Output data buffer Referring now to FIGS. 5 and 6 an output data buffer 70 is generally indicated by block 501. All of the output data buffers are substantially identical with the exception that the first output data buffer has minor changes therein as described hereinafter. Readout by data output buffer 501 is initiated by 75 a pulse on terminal ST? which pulse is supplied by the

17 prior output data buffer. In the event that the output buffer is the first buffer, terminal STP extends to the corresponding terminal in the common control which, as previously described, pulses tern-,Linal STP to initiate the readout of the first output data buffer when the system is synchronized. In any event the pulse on terminal STP passes by way of lead 515 to the SET input of MS flipflop 502 thereby setting the flip- flop. This occurs upon the initiation of the clock pulse whereby MS flip-flop 502 is SET concurrently with the reception of the initiation of the first bit of the code character to be read. With MS flip-flop 502 SET, the "I" output terminal thereof goes higb, and this high condition is applied to one input lead extending to AND gate 507. Lead 519 also extends to gate 507 and lead 519 in turn is strapped to positive battery in all output buffers with the exception of the first buffer. In the event that this is the first buffer, lead 519 is strapped to terminal DIS. As previously described, a high condition is applied to terminal DIS by the output common control when the system is synchronized, and the operation of the first output data buffer is enabled. In any event, assuming a normal operation, a high condition is applied to lead 519 whereby gate 507 is enabled. The third input to gate 507 extends to the "not" clock terminal. Accordingly, the "not" clock pulses are applied through gate 507 to AND gate 508 which pulses occur at the theoretical midpoints of the bits of the data character. Returning now to the setting of MS flip-flop 502, the high condition transition -at output terminal "I" is also applied through lead 503 to- monopulser 570 which in turn applies a pulse to one input of gate 504. As described- hereinafter, CM flip-flop 512 is in the CLEARED condition so long as output data buffer 501 is prepared to register a received character. With CM flip-flop 512 in the CLEARED condition, the "O" output thereof applies a high condition to gate 504. Accordingly, upon the setting of MS flip-flop 502, monopulser 570 passes a pulse thtough gate 504 to clear SD flip-flop 505. SD flip-flop 505 in tum applies a high condition to lead 506 which lead extends to one input AND gate 508. Lead 518 also extends to gate 508, and lead 518 in turn is connected to terminal DD. As previously described the output common control applies a high condition to terminal DD when the system is synchronized and the output data buffers are permitted

to register characters. Accordingly, assuming the system is synchronized and a high condition is applied by way of lead 519 to gate 508, the clearing of SD flip- flop 505 enables gate 508 to pass the "noV' clock impulses provided by gate 507 to the shift pulse input of the bus sbift register generally indicated by a block 511. Accordingly, with output data buffer 501 prepared to read in a,data character, the "not" clock pulses are passed through gate 508 to provide shift pulses for bus register 511. It is noted that the "not" clock pulses passed through gate 507 are also applied to the count input of counter 509, counter 509 normally providing a low condition at its output to gate 510. When counter 509, however, counts up to a number corresponding to intelligence elements of the code character plus 1, the output thereof goes to the high condition. Accordingly, counter 509 provides a count corresponding to the number of intelligence elements plus the flag bit of the code character. Returning now to shift register 511, it is noted that the shift register includes a plurality of stages designated 1 through N which stages- correspond to the N intelligence elements of the code character and stage F which corresponds to the flag bit -accompanying the code character. The incoming code character is applied to terminal BO of output data buffer 501 from the system bus. Terminal BO in turn is connected through lead 517 to stage F of shift register 511. Accordingly, at the theoretical midpoint of the first data bit applied to stage F by the 3,466,397 18 common bus, the "not" clock impulse applied through gate 508 drives stage F to the condition corresponding to this first bit. At approximately the theoretical midpoint of the second bit, the "not" clock impulse applied through gate 508 shifts the first bit to stage N and drives stage F to the condition corresponding to the second bit. In a similar manner each subsequent bit of the code character is inserted in shift register 511, and the prior bits are shifted down through the stages untfl the first bit 10 is stored in stage 1, the last bit is stored in stage N, and the flag bit is stored in stage F. The "not" clock pulse which applies the flag bit to stage F also advances counter 509 to its final count. Accordingly, counter 509 applies a high cdndition to gate 15 510 enabling the gate. With gate 510 enabled, the next clock pulse is passed therethrough to clear MS ffip-flop 502. This removes the high condition from gate 507 and the gate is diabled. Accordingly, the "noC' clock pulses are now blocked, the advancing of the counter 509 is 20 stopped, and the registration of subsequent code elements from the common bus is terminated. The clearing of MS flip-flop 502 also drives the "Oll output thereof to the high condition. This transition to the high condition is passed by way of lead 516 to ter- 25 minal STS. As previously described, terminal STS is connected to the terminal STP of the subsequent output data buffer whereby the reading of the subsequent data buffer is initiated. Of course, in the event that output data buffer 501 is the last buffer, then the high condition transition 30 on lead 516 is passed by way of terminal STS to the output common control to enable it to scan the framing bit as previously described. With the high condition provided at the "O" output terminal of MS flip-flop 502, a reset pulse is passed to 35 counter 509 and counter 509 is reset to its initial count. In addition, the high condition at the "O" output of MS flip-flop 502 is passed through lead 513. 'Ehis high condition transition is thus applied to the SET input of SD flipflop 505, and the setting of SD fflp-flop 505 removes the 40 previously described application of the enabling potential to gate 508. The high condition on lead 513 is also applied to one input of AND gate 520. The other input to AND gate 520 extends to lead 523. As described hereinafter, lead 45 523 is normally iii the hiah condition with the exception of the situation wherein "idle" characters are being registered by shift register 55. Accordingly, assuming that shift register 511 is not registering an "idle" character, with lead 523 in the high condition and lead 513 going to the 50 high condition, gate 520 is enabled to set CM flip-flop 512. Accordingly, upon the registration of the character in shift register 511, CM flip-flop 512 is set, its "O" output terminal goes low and gate 504 is disabled. When CM flip-flop 512 is SET, the "I" output terrninal 55 Of CM flip-flop 512 goes high enabling gate 521. The other input to gate 521 extends to lead 524. As described hereinafter, lead 524 is in the high condition when the output buffer is not outpulsing a data character which condition permits the readout of the character in bus shift register 60 511. Accordingly, assuming that the readout of shift register 511 is permissible and a high condition is applied to lead 524, AND gate 521 pulses monopulser 522. Monopulser 522 in response provides a gate pulse at the output thereof. 65 The gate pulse at the output of monopulser 522 is passed to lead 525, and then to an input of gate 526. The other input to gate 526 is connected to lead 527.

Lead 527 is normally in the high condition with the exception of the situation wherein shift register 511 is storing a char- 70 acter representing the "break" or prolonged space condition. Assuming a "break" character is not stored in sbift register 511, the high condition on lead 527 enables gate 526 to pass the gate pulse therethrough. The output of gate 526 is connected to the CLEAR inputs of stages I through 75 N and to the SET inpiit of stage F in shift register 511.

19 Accordingly, the pulse passed through -ate 526 clears stages I through N and sets stage F. The insertion of marking bits in stages I through N and a spacing bit in stage F corresponds to the "idle" character. Accordingly, upon the readout of shift registetr 511, as described hereinafter, the "idle" character is inserted therein. The gate pulse provided by monopulser 522 is also applied by way of lead 525 to a readout gate generally indicated by block 601, FIG. 6. Gate 601 functions to read out tile character stored in bus shift register 511 into the channel shift register generally indicated by block 602. in addition, the gate pulse on lead 525 passes to lead 604 which is conected to the SET input of stage STP in channel shift register 602. Accordingly, the gate pulse inserts a spacing bit in stage STP corresponding to the spacing start element of the code character. In addition, the gate pulse at the output of monopulser 522 is passed to the CLEAR input of flip-flop 512. The consequent clearing of CM flip-flop 512 reenables -ate 504 as previously described, thus indicatidg that the character in bus shift register 511 has been read out. Returning now to gate 601, it is noted that the @ate includes AND gates 605 thr6ugh 609, and AND @ates 611 through 615. Gates 605 through 607 have one input thereof connected by way of leads 530 through 532 to the "W' otitput terminals of stages I through N of shift register 511. The other input to gates 605 through 607 extends to lead 525. Since the outputs of gates 605 through 607 are connected to the CLEAR inputs of stages I through N of channel register 602, upon the application of the gate pulse to lead 525, gate 601 reads out the marking elements in bus shift register 511 and inserts them into channel shift register 602. Similarly, one input of gates 611 through 613 in gate 601 is conected to the "1" outputs of stages 1 through N in bus re-ister 511 by way of leads 535 through 537. With the other input to gates 611 through 613 connected to lead 525 and the output of gates 611 through 613 connected to the SET inputs of stages I throu@-h N of channel register 602, these gates, in response to the gate pulse, read out the spacin.- elements in bus register 511 and insert them iii channel register 602. The gate pulse on lead 525 also functions with gates 608, 609, 614 and 615 to insert the parity bit of the code character in stage P and the final or stop bit of the code character in stage SP of channel shift register 602 as described hereinafter. The insertion of data bits in channel shift register 602 initiates the operation of the outpulser circuit to transmit the data to output lead 628. The sensing of the character in shift register 602 is performed by OR gate 620. The input leads to OR gate 620 are connected to the "O" output terrninals of stages 1 through N, P and SP of shift register 602. Assuming now that gate 601 reads out a character from bus shift register 511 into channel shift re-ister 602 as previously described, a marking bit will be applied to one of the stages 1 through N, P or SP of shift register 602. - Accordingly, a "O" output terminal of one or more of the stages will apply a high condition to OR gate 620 which high condition is applied therethrough to oscillator control 621. Oscillator control 621 operates in response to the application of the high condition thereto to enable oscillator 622. Oscillator 622 in response thereto provides at the output thereof a train of pulses at a bit rate corresponding to the signal rate on data output lead 628. The first output pulse of oscillator 622 is concurrently applied to the SET lead of CG flip- flop 623 and to an input lead of OR gate 624. At this moment CG flip-flop 623 is normally CLEAR whereby thp. "O" output thereof applies a high condition to the other input of OR gate 624. Accordingly, OR gate 624 normally applies a high condition to the input of monopulser 629. The "O" output terminal of CG flip-flop 623 is also connected by way of delay network 627 to lead 524. With CG flip-flop 623 normally in the CLEARED condition, the high condition 3,466,397 20 at the "O" output terminal thereof is passed through delay network 627 to lead 524 whereby, as previously described, -ate 521 is enabled to operate monopulser 522 to provide the gate pulse to read out bus shift re.-ister 511. When oscillator 622 sets CG flip-flop 623, however, th-hi.-h condition at the "O" output tcrminal thereof is removed, removing the high condition applied to lead 524. Accordingly, during the outpulsin.- of the code character, gate 521 is disabled. 10 The first OUtpUt PLI]SE of oscillator 622 sets CG flip-flop 623

thereby removing the high condition at the "Ol' OLITput thereof. Thus, gate 521 is disabled and the high condition applied throu@-h gate 624 to monopulser 629 is removed. At this time, however, oscillator 622 is applyin@- 15 the first output pulse to OR -ate 624. Thus, the outplit of OR -ate 624 remains hioh and the firs' output p-Lilse of oscillato@- 622 does not provide a positive transition to monopulser 629 whereby monoptilser 629 is not operated. It is noted that at this time that data output lead 623 20 which is connected to the "O" output terminal of sta-e STP is in the low condition, simulating a spacing sta@rt signal, since the prior gate pulse applied by way of lead 604 has set stage STP. At the conclusion of an element duration, oscillator 25 622 provides the second olitput pulse. This second pulse is passed through OR @ate 624, and monopulser 629, in response thereto, provid'es a pulse to lead 625. Lead 625 is connected to the shift pulse input of channel shift register 602 and to the SET input of sta@e SP by way of OR 30 gate 626. Accordingly, at the termina'tion of the start bit interval, monopulser 629 applies a shift pulse to shift register 602 and concurrently inserts a spacing bit in stage SP. The application of the shift pulse ftinctions to advance all the cod-- elements one stage, thereby shifting the code 35 element stored in the first stage of shift register 602 to stage STP. Accordingly, the first intelligence element of the data character is applied to data output lead 628. In a similar manner, each subsequent pulse from oscillator 622 operates monopulser 629, and monopulser 40 629 in turn advances the data bits through the sta,-es in shift register 602 whereby successive data bits are shif@ed to stage STP and thus provided to data output lead 628. Concurrently therewith, each shift pulse on lead 625 is applied through OR gate 626 to insert spacing bits in 45 stage STP. Thus, shift register 602 fills up with spacinbits which follow the data character down the shift re,@is@ ter. The application of shift pulses to shift register 602 continues until the stop bit previously stored in stage SP 50 is shifted to stage STP. At this moment all the other stages are filled with the spacing bits. Accordingly, all of the "O" output terminals thereof are in the low condition and OR gate 620 provides a low condition at the output thereof. This low condition removes the enabling potential from 55 oscillator control 621, and oscillator 622 is th--reby disabled. The low condition at the output of OR gate 620 is also applied to inverter 630 which in turn passes a bigh condition to the CLEAR input of CG flip-flop 623. Accordingly, CG flip-flop 623 goes to the CLEAR condition, 6o reapplying a high potential to OR gate 624. In addition, the high condition at the "O" output terminal of flip-flop 623 is applied to delay network 627. Delay network 627 delays the passa@e of the high condition therethrotigh for an interval corresponding to the duration of the stop ele- 65 ment. At the termination of this interval, the hi-h condition is passed through to lead 524 whereby AND gate 521 is a.-ain enabled. This indicates that the outpulsing of the data character has been completed arranging the circuit to permit the generation of another gate pulse by 70 monopulser 522. Su-mmarizing the outpulsing operation, when a gate pulse generated by monopulser 522 reads a cbaracter into channel shift register 602, characters are detected by OR gate 620 to start the outpulser eirCLlit which CirCLlit in- 75 cludes oscillator"control 621, oscillator 622 and CG flip-

21 flop 623. In response to the detection of the character, the outpulser circuit provides shift pulses to channel shift register 602 whereby each of the code character bits is presented to data output lead 628. In addition, the shift pulses function to insert spacing bits in shift register 602 thereby filling up th-. shift register as the data character is shifted out. Concurrently with the outpulsin- of the data character, AND gate 521 is disabled thereby precludin.- the operation of monopulser 522 to generat6 another -atin-, pulse. Accordingly, during the outpulsing operation, a subsequent character cannot be read from bus shift register 511 into channel register 602. When the code character is fully read out of channel shift register 602, the outpulser circuit is restored to the idle condition, the shift pillses are terminated, and AND gate 521 is reenabled to permit the subsequent generation of another gate pulse. Assuming now that a character is read into bus register 511 and at the conclusion thereof CM flip-flop 512 is SET, as previously described, CM flip-flop 512 in response thereto provides a positive transition to one input of gate 521. If, at this time, a code character is being outpulsed, gate 521 is disabled, as- previously described, and monopulser 522 is therefore not enabled to generate the gate pulse. CM flip-flop 512, -however, remains SET maintaining the high condition on gate 521. Accordingly, at the termination of the outpulsing, -a high

condition is applied to lead 524, as previously described, whereby monopulser 522 is operated and the gate pulse is generated to transfer the new character from bus register 511 to channel register 602 in the same manner as previously described. Assuming now that a code character is being outpulsed out of channel register 602 and another code character is stored in bus register 511 awaiting transfer to channel register 602, the registration of a new character in bus register 511 is precluded even though a new start-to-scan cycle is initiated by the application of a pulse to terminal STP by the prior output data buffer. With the character stored in bus register 511, SD flip-flop 505 is SET, as previously described. Accordingly, a low condition is applied from the "O" terminal of SD flip-flop 505 to lead 506 to one input of gate 508 thereby disabling the gate. In addition, CM flip-flop 512 is SET as previously described, and since the generation of a gate pulse is precluded, the flip-flop remains SET so long as the character in channel register 602 is being outpulsed. Accordingly, the "O" output terminal of CM flip-flop 512 applies a low condition to gate 504 disabling the gate. Upon the application of the new start-to-scan signal to terminal STP, MS ffip-flop 502 is set, as previously described. This enables gate 507 and applies a high condition to gate 504. Since CM flip-flop 512 remains SET, however, gate 504 is disabled, precluding the clearing of SD flip-flop 505. Accordingly, gate 508 remains disabled. Thus, the "noe' clock impulses are passed through gate 507 but blocked by gate 508. This precludes the entering of the character in bus register 511 but permits the counting of the bits by counter 509. Accordingly, although the registration of the character is precluded, the counter counts the data bits and at the conclusion thereof, MS flip-flop 502 is CLEARED as previously described thereby sending the start-to- scan signal to the next output data buffer. Accordingly, the sequential read-in of the characters by the output data buffers is continued although, with a character stored in bus register 511, one of the data characters which is presumably an "idle" character is discarded. Detection of code and flag bits Gate 601 in addition to inserting the data character in channel register 602 also gates the parity bit and the stop bit into stages P and SP of the channel register. The bit gated into stage P corresponds to the parity bit of the code character provided to the input data buffer. Similarly, the bit inserted in stage SP comprises the stop bit and 3,466,397 22 corresponds to the stop bit of the code character applied to the corresponding input data buffer. Assuming now that the code character applied by way of the bus to bus register 511 corresponds to an idle condition, this "idle" character, as previously described, supplies marking bits to stages I through N and a spacing bit to stage F in bus register 511. AND gate 540 has a plurality of inputs connected to the "O" output terminals of stagps I through N, which terminals go to the high 10 condition when the corresponding stage stores a marking bit. Accordingly, the insertion of the "idle" character in bus register 511 enables AND gate 540 to produce a high condition at the output thereof. This high condition is provided to AND gate 541. The other input to AND gate 15 541 extends through lead 538 to the "1" output terminal of stage F of bus register 511. Since the "I" output terminal is also in the high- condition, gate 541 passes a high condition to inverter 542. Inverter 542 in turn applied a low condition to leqd 523. With a low condition on lead 20 523, gate 520 is disabled, precluding the SETTING of CM flip-flop 512. Accordingly, the generation of a gate pulse is precluded, th@ insertion of a new character in channel register 602 is blocked, the operation of the outpulser circuit is thus not initiat-@d, aiid the markin@. stop 25 bit in stage STP is retained, thereby maintaining data output lead 628 in the idle marking condition. When a "letters" character is received from the bus, marking bits are ins-,rted in stages I through N and a markidg llag bit is inserted in stage F of bus re.-ister 511. 30 With marking bits in stages 1 through N, a high condition is produced at the output of gate 540 in the same manner as previously disclosed for the situation wh-.n an "idle" character is received. Since a marking flag bit is received for the "letters" character, however, a low condition is 35 provided by way of lead 538 to gate 541. Accordingly, inverter 542 provides a high condition to lead 523. This enlables gate 520, permitting the setting of CM flip-flop 512 whereby monopulser 522 can provide a gate pulse. Accordingly, the registration of the "letters" character per- 40 mits the generation of a gate pulse and the readout of bus register 511 into channel register 602. As previously disclosed, the code characters include parity bits. Assuming even parity and an odd number of information bits or odd parity and an even number of 4,,@ information bits, the "letters" character requires a marking parity bit. In this event, the output of gate 540 is strapped to one ir@put of AND

gate 545. Thus, the high condition at the output of gate 540 is passed through OR gate 545 and then by way of lead 546 to AND gate 608. 50 Since the other input to AND gate 608 extends to lead 525, the gate pulse is thus passed through gate 608 to the CLEAR input of stage P of channel register 602. Accordingly, a marking parity bit is inserted in stage P. Conversely, if the parity bit for the "letters" character 5 - of the code dedicated to output data buffer 501 is spacing, the connection of gate 540 to gate 545 is open. Accordingly, OR gate 545 applies a low condition to inverter 549 and inverter 549 in turn applies a high condition by way of lead 550 to gate 614. The gate pulse is therefore 60 passed by gate 614 to the SET input of stage P whereby a spacing parity bit is inserted in channel re-ister 602. With the "letters" character stored in bus' register 511, the output of AND gate 552 is in the low condition, as described hereinafter, whereby the output of gate 553 is 65 similarly in a low condition. This low condition is provided to inverter 554 and inverter 554 in turn applies a high condition to lead 566. Lead 566 extends to an input of gate 609 enabling gate 609 to pass the gate pulse theretbrough to the CLEAR input of stage SP of channel 70 register 602. Accordingly, a marking stop pulse is inserted in stage SP. The high output of inverter 554 is also passed to lead 527 enabling gate 526 whereby upon the readout of bus register 511 the "idle" character is reinserted therein, 75 as previously described. Accordingly, the "letters" char-

3,466,397 23 acter is read out of blis register 511 into channel register 602, the appropriate parity bit is inserted in stage P and a marking stop bit is inserted in stage SP. Since the gate pulse inserts a spaciiig star-t bit in stage STP and marking bits are inserted in channel register 602, the operation of the outpulsing circuit is initiated and a start-stop "letters" character is applied to data output lead 628. When a "break" character is received by bus register 511 data outptit lead 628 is placed in the spacin- condition. This condition is maintained until a character other than the "break" character is applied to btis register 511. It is recalled that a "break" character comprises all spacing information bits and a marking flag bit. Accordingly, wben a "break" character is received, stages I throu-,h N of bus register 511 are SET and stage F is CLEARED. The "I" output terminals of stages I tlirough N of bus register 511 are connected to the ini)uts of AND gate 552. Since all the "1" output terminais are in the high condition when a "break" character is received, the output of AND gate 552 goes hi.-h. This high condition is applied to inverter 562 which in ti-irn applies a low condition to gate 548 thereby disablin- the gate. With a low condition provided to the output of gate 548, the output of OR - --ate 545 is lo@v, and inverter 549 provides a high condition to lead 550. Since lead 550 extends to one inptit of -ate 614, -ate 614 is enabled to pass a -ate piilse tberethrough to SET stage P of channel register 602. Thiis, the storage of a "break" signal in bus register 511 resiilts in the insertion of a spacing bit in stage P. In addition, the output of @ate 552 is connected to one input of gate 553. The other input to -ate 553 is connected by way of lead 533 to the "O" out'put terminal of sta.-c F in biis register 511. Accordin@ly, botli inputs to gate 553 are high, and gate 553 in tum applies a hi,-h condition to the otitput thereof. The hi,-h cond;tion at the outptit of gate 553 is passed by way of lead 560 to AND -ate 615 in gate 601. Accordingly, when a @ate plilse is subsequently produced, this gate pulse passes through gate 615 and OR gate 626 to the SET inptit of stage SP in channel register 602. Thus, the storage of the "break" signal in bus register 511 results in the insertion of a spacing bit in stage SP of channel register 602. The high condition at the olitput of @ate 553 is also applied to inverter 554 which in turn passes a low condition to lead 527. This low condillion on lead 527 disables gate 526. With gate 526 disabled, the gate pulse cannot insert the "idle" character in pulse register 511. This maintains the "break" condition if synchronism is lost, as described hereinafter. The high condition at the output of @ate 553 is also applied by way of lead 556 to one input of -ate 557. With the other input of gate 557 connected to lead 525, the gate pulse is enabled to pass through gate 557 to clear SS flip-flop 558. As described hereinafter, SS flipflop 558 remains in the CLEARED condition so long as the "breav' characters are bein.- received, Assuming now that the outpulser circuit is prepared to accept the "break" character, gate 521 is enabled. After the "break" character is received, CM flip-flop 512 is SET in the same manner as previously described whereby AND gate 521 applies a high condition to monopulser 522. Accordingly, monopulser 522 produces the gate pulse and the "break" character is read from bus register 511 into channel re.-ister 602. Since, as previously describ,d, all

the intelligence bits are spacing, and spacing bits are inserted in stages P and SP of channel re.aister 602, OR ,-ate 620 does not read any rnarking bits, and the operation of the outpulser circuit is not initiated. The gate pulse, however, has SET stage STP in channel register 602. Accordingly, data output lead 628 is placed in the spacing condition, which condition is maintained to simulate a "break" condition. This operation is repeated for each successve reception of the "break" signals whereby 24 the spachig condition of data otitput lead 628 remains undisturbed. At the termination of the "break" condition, a character other than the "break" character is received by bus register 511. Accordingly, the output of gate 552 goes dovin and the output of gate 553 correspondin,-Iy goes down. This restores the low condition to leads 556 and 560. In addition, inverter 554 restores the high condition to lead 527 and with MS flip-flop 502 being CLEARED 10 at the termination of the new character, lead 513 goes high whereby a high condition is passed by gate 564 to the SET input of SS flip-flop 558. This restores SS flipflop 558 to the SET condition, providing a positive transition at its "1" output terminal. This positive transition is 15 passed by way of lead 559 to the CLEAR input of stage STP in channel register 602. Accordingly, upon the reception of a character other than the "break" signal, stage STP is CLEARED and data output lead 628 is restored to the marking condition, thus terminating the "break" 20 si.,nal. The "blank" character, when received by the output data.buffer, comprises all spacing inforination bits and a spacing fla.- bit. Accordin.-ly, when a "blank" character is received, stages I throuah N of bus register 511 are 25 SET, and stage F is SET. Since all the "1" output terminals of stages I through N are in the high condition, the output of AND gates 552 goes high. With stage F set, however, the "O" output terminal thereof applies a low condition to gate 553 by way of lead 533. The output 30 of gate 533 therefore goes low. In addition, the low condition on the "O" output terminals of stages I through N in bus register 511 drives the output of gate 540 to the low condition, disablin@ @ates 541 and 544. Inverter 542 thus applies a high condition to lead 523. This permits 35 the gencratiori of the gate pulse to transfer the "blank" character to channel register 602. Finally, the low condition at the output of gate 553 is passed to inverter 554 which in turn provides high conditions to leads 527 and 566. With a high condition on lead 566, the gate pulse 40 inserts a markin- stop bit i-n stage SP of channel register 602. Accordingly, the "blank" character is read out of bus register 511 and inserted in channel register 602 in the conventional manner, the gate pulse inserting the appropriate start and stop bits. We have previotisly assumed that the code character 45 includes a parity bit which provides even parity. With this arrangement inverter 562 is connected to gate 548 whereby gate 548 is disabled -apon the reception of the "blank" character. With both -ates 544 and 548 disabled, OR gate 545 applies a low condition to inverter 549. In- 50 verter 549 in turn applies a high condition to lead 550 enabling gate 614 whereby a spacing bit is inserted in stage P of channel register 6.02. Accordingly, with the even parity code, a spacing parity bit is inserted upon the recepti(yn of the "blank" character. 33 As previously described, the input data buffer during the transmission of normal data characters provides a spacing flag when a marking parity bit is received and a marking flag when a spacing parity bit is received. The output data buffer then examines the flag bit and inserts 60 the appropriate parity bit in sta,@e P of channel register 602. During the reception of normal characters, the output of gates 540 and 552 are low, and the normal transfer of characters from bus register 511 to channel register 602 is provided as previously described. With 65 gate 552 low, inverter 562 provides enabling potential to gate 548. Concurrently, with the output of gate 540 low, gate 544 is disabled. - Accordingly, only gate 548 can apply a high condition to OR gate 545. io Assuming now that the received code character includes a marking flag, the "I" output terminal of stage F in bus register 511 goes low, and gate 548 is disabled. The resultant low condition at the output thereof is applied to inverter 549 and inverter 549 in turn applies a high condition to lead 550. Accordingly, gate 614 is en- 15 abled and a spacing bit is inserted in stage P of channel

 register 602. Conversely, if a spacing flag is received, the "1" output of stage F of bus register 511 goes high, and gate 548 applies a high condition through OR gate 545 to lead 546. This enables gate 608 to insert a marking bit in stage P of channel register 602. Accordingly, upon , , the reception of a marking flag, a spacing parity bit is a inserted and upon reception of a spacing flag, a marking parity bit is inserted in channel register 602. During the reception of

normal characters, the circuit also inserts a stop bit into channel register 602. When a 10 conventional character is received, the output of gate 552 goes low as previously described. The output of gate 553 consequently also goes low and inverter 554 applies a high condition to lead 566. Gate 609 is thus enabled to pass the gate pulse therethrough. Accordingly, a marking 1,5 bit is inserted in stage SP which bit constitutes the mark. ing stop element of the code character. Bit distribution during synchronization recovery When synchronization is lost, the output common 20 control precludes the registration of characters and the counting of character bits by the output data buffers as previously described. When a proper framing bit is de- tected, however, the output data buffers are permitted to count the data bits but the registration of the characters 25 is precluded until three successive proper framing bits are detected. Assuming now that the system loses syn chronization, the normal high conditions on terminal DD is removed. In addition, the high condition applied to terminal DIS 30 in the first output data buffer is also removed. Accord- ingly, gate 508 in each of the data buffers is disabled, and gate 507 in the first output data buffer is similarly disabled. With gate 507 disabled in the first output data buffer, the "not" clock pulses applied therethrough to 35 counter 509 are blocked. When a scan cycle is started by the output cammon control, the first output buffer MS flip-flop 502 is SET, as previously described. During loss of synchronization, however, gate 507 is disabled blocking ,the application of the "not" clock pulses to counter 509. 40 Thus MS flip-flop 502 remains SET while the low condi- tion is maintained on terminal DIS. When, after losing . synchronization, an appropriate framing bit is detected, the condition on terminal DIS goes high, and gate 507 in the first output data buffer 45 is enabled. Thereafter the "not" clock pulses are permit- ted to pass through gate 507 to counter 509. Gate 508 remains blocked, however, because of the low conditions on terminal DD, precluding the registration of the char- a,cter received from the bus on lead 517. - AccordinglY, 50 although the character is not registered, at the termination of the appropriate count MS flip-ffop 502 is CLEARED, as previously described, and the start-to-scan signal is sent to the second output data buffer. When the second output data buffer receives the 55 start-to-scan signal on its terminal STP, the MS flip-flop 502 therein is set, enabling gate 507. The DD terminal, however, is still in the low condition, and gate 508 is disabled. Accordingly, the second output data buffer counts the data 4bits, but does not register the data character. 60 Thus, each output data buffer counts the bits and starts the next data buffer but does not register the character. The process is repeated until the last buffer counts the bits and, at the termination of the count, the last data buffer signals the output common control over its terminal STS-N and 65 the output common control scans the next bit to determine if it is the proper framing bit as previously described. These cycles are repeated until the output 'common control regains synchronization, as previously described, restoring the high conditions on terminals DIS and DD on the out- 70 put data buffers. It is noted that during loss of synchronization, when the output data buffers are counting but not registeridg the characters, MS fflp-flop 502 therein cycles by being successively SET and CLEARED. This, in turn, suc- 75 26 cessively CLEARS and SETS SD flip-flop 505. CM flipflop 512, however, remains in the CLEAR condition, since, as previously described, the circuit inserts the "idle" character in bus register 511 whereby a low condition is provided to lead 523. This low condition on lead 523 disables gate 520, precluding the setting of CM flip-flop 512. Accordingly, upon loss of synchronization, each output buffer inserts the "idle" character in bus register 511, precluding the outpulsing of channel register 602 whereby data output lead 628 is maintained in the idle marking condition. In the event, however, that a "break" character is in bus register 511, CM flip-flop 512 cycles since lead 527 is maintained in a low condidon, as previously- described. This low condition disables gate 526 whereby the "idle" character is not inserted in bus register 511. Accordingly, the "break" character is retained in bus register 511 as previously described. With the "break" character retained in bus register 511, outpulsing of channel register 602 is precluded, as previously described. A spacing "break" condition is maintained on output lead 628, however, since stage STP of channel register 602 has been SET, as previously described. Thus, if the output data buffer received a "break" signal just prior to loss of synchronization, the "break" condition on data output lead 628 is maintained until synchronization is restored and a new character is received. Although a specific embodiment of the invention has been shown and described, it will be understood that various modifications may be made

without departing from the spirit of the invention and within the scope of the appended claims. What is claimed is: 1. In a time division system for transmitting data codes which include permutation code element characters, a common transmission path, a plurality of sequential input ports, each of said input ports being dedicated to a predetermined one of said codes, a plurality of output ports, each of said output ports associated with an input port and dedicated to the corresponding code, signalin.- means individual to each of said input ports for sending a code character to said common path, said sigr@aling means including means for serially applying a plurality of said code elements to said common path corresponding to the number of elements in each of the characters of the code dedicated to said input port, means responsive to each of said signaling means applying the last element of said plurality of elements to said path for initiating the operation of the signaling means individual to the next subsequent one of said input ports, receiving means individual to each output port for receiving a code character from said path, said receiving means including means for reading a plurality of serial elements applied to said path corresponding to the number of elements in each of the characters of the code dedicated to said output port, and means responsive to each of said receiving means reading the last element of said plurality of elements for initiating the operation of the receiving means individual to the next subsequent one of said output ports. 2. In a time division transmission system, a plurality of sequential input ports for receiving code character bits, an output port corresponding to each input por@t, a common transmission path interconnecting said input ports and said output ports, means individual to each of said input ports for serially applying aR of the bits of said received code cha@racter to said path, input port enabling means responsive to the application

3,466,397 27 of all of the bits of the character to said path by each of said applying means for enabling said applying means individual to the next subsequent one of said input ports, means included in each of said output ports for registering all of the bits of a code character applied thereto by said path, and output port enabling rneans individual to each of said re-istering means and responsive to the appli- cation of all of the bits of the character thereto for 10 enabling the application of bits from said path to said registering means individual to the next subsequent one of said output ports. 3. In a time division transmission system in accordance with claim 2 further including a source of pulses having ;5 a repetition rate corresponding to the signaling rate of said path, said applying means including means enabled by each pulse from said source for applying a bit to said path, said input port enabling means including a pulse steering circuit for steerin.- circuit for steering said Pulses 20 from said source to the next subsequent one of said input ports. 4. In a time division transmission system in accordance with claim 3 wherein said registering means includes means enabled by each pulse from said source for register- 25 ing a bit applied by said path, and said output port en- abling means includes a pulse steering circiiit for steering said pulses from said solirce to the next subsequent one of said output ports. 5. In a time division transmission system in accordance 30 with claim 2 wherein said applyin.- means includes a storage circuit for storing said bits of said received char- acter prior to said application to said path, and said inpi-it port enabling means includes means for detectin,@ the pres- ence of said bits in said storage circuit, said - detecting 35 means including means responsive to the removal of all of said bits from said storage circliit for enabling said next subsequent input port. 6. In a time division transmission systeni in accordance with claim 5 wherein said applying means includes means 40 responsive to said enabling of said applying means for inserting a ffag bit in said stora,@e circuit for application to said path. 7. In a time division transmission system in accordance with claim 6 wherein said inserting means includes means 45 operable in the absence of the reception of a code char- acter by said input port for inverting the flag bit. S. In a time division transmission system in accordance with claim 7 wherein said re.-istering means includes means responsive to the registration of said inverted r)o flag bit for applying a prolonged si-nal condition to said output port. 0 9. In a time division system in accordance with claim 2 wherein each of said output port enabling means in- 28 ing means, said counting means arranged to operate said output port enabling means upon counting a predetermined number of bits corresponding to the number of bits included in said code character. 10. In a time division system in accordance with claim 9 further including synchronizing means for conctirrently

enablin.- said applying means individual to one of said input ports and registering means individual to the corresponding one of said output ports, and means for detecting failure of said synchronizing means, said failure detecting means further including means for disabling said counting means and said registering means. 11. In a tirne division system in accordance with claim 10 wherein said failure detecting means includes means for recovering synchronization, said synchronization recovering means further incltiding means for reenabling said disabled counting means whereby each of said output ports provides counting but precludes bit registration. 12. In a time division signaling system which includes a data signal repeater circuit arranged to read the signal element conditions of incoming code characters and generate and sequentially apply bits to an output path, said bits corresponding to the signal conditions of the code character elements, said repeater circuit being further arranged to generate and apply a flag bit to the output path, and means responsive to the reception of a prolonged signal condition for modifying the flag bit, whereby the sequence of bits generated for the prolonged signal condition is distinguishable from the sequence of bits-generated for the code characters having all elements corresponding to the prolonged signal condition. 13. In a time division signaling system in accordance with claim 12 wherein said signal elements of said code characters include a parity signal element, said repeater circuit being flirther arranged to normally generate the flag bit to correspond to the parity signal element condition and said modifying means being arran,-ed to invert the signal condition of the fla.- bit. References Cited UNITED STATES PATENTS 3,197,563 7/1965 Hamsher ---------- 179-15 3,310,626 3/1967 Cassidy ---------- 179-15 3,334,181 8/1967 Bartlett ---------- 179-15 3,366,737 1/1968 Brown ---------- 179-15 XR 3,377,585 4/1968 Magnin ---------- 179-15 2840,705 6/1958 Scully ---------- 340-147 XR 2,919,435 12/1959 Hawley ---------- 340-147 3,065,303 11/1962 Kaneko ---------- 179-15 RICHARD MURRAY, Primary Examiner cludes means for counting the bits applied to said register- 55 CARL R. VON HELLENS, Assistant Examiner

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May 6, 1969 P. BENOWITZ ET AL 3@443,022 HUB COUPLING SYSTEM Filed Aug. 2, 1965 tn tc, qo OD CO tn izk Qk < tu 'mF j@l kZ, c@F Q:i 44 CY Hi- @4 tu tu 4i 4t @4 P. BEIVOWITZ INIIENTORS H. KAHL BROCK ATTORNEK

3 1 4 4 3 @ 0 2 2 United States Pitent Office Patented May 6, 1969 2 It is a feature Gf this invention that the double space 3,443,022 detector is provided on a per hifb basis common to all HU@ COUPLING SYSTEM the subsrribers rather than individual tD each subscriber. Paul Benowitz and Heinz KahIbrock, Brooklyn, N.Y., as. It is a feature of this invention that the common detec- signors to Bell Telephone Laboratories, Incorporated, tor unblocks all the sending legs when a double space is New York, N.Y., a corporation of New York 5 FiIed Aug. 2, 1965, Ser. No. 476,515 detected. Int. Cl. HOII 15100; H03k 5120 It is a feature of this invention that a tim@d "break" U.S. Cl. 178-2 8 Claims signal is applied to the hub when a double space is detected. This advises all subscribers that the sending io subscriber has been interrupted. In addition, since the ABSTRACT OF THE DISCLOSURE Subscriber data

lines are interconnected by way of a common hub. To preclude the recirculation of signals from the subscriber receiving leg back to the sending leg, 15 each subscriber is provided with a control circuit which blocks the retransmission of signals from the hub to the sending leg while data signal-s are being received from the subscriber. To advise all subsctibers that the sending subscriber is being interrupted by another subscriber, a "double space" detector common to all the subscribers 20 detects when two or more send into the hub and in re- sponse thereto unblocks all the sending legs and seiids a time "break" signal into the hub and there'fore to all sending legs. 25 This invention relates to a data channel hub concen- trations and, more particularly, to control circuits for hub coupling circuits. It is a broad object of this invention to provide an 30 improved control circuit for hub coupling units. Hub concentrations provide arrangements for permit- ting any one of a group of data exchange subscribers to transmit to all others in the group. This is accomplished by connecting each subscriber transmission circuit to a 35 common receiving hub or terminal by way of an individ- ual receiving leg and to a common sending hub by way of an individual sending leg. By interconnecting the re- ceiving hub and the sending hub, by way of a regenerative repeater, for example, th6 marking and spacing data 40 signal,s incoming from any one of the subscriber trans- mission circuits are applied to the receive hub via the individual receiving leg, repeated to the send hub, and thus simultaneously transmitted to all other subscribers through their respective sending legs. 45 When a subscriber is sending into the hiib, provision must be made to preclude retransmission back to the sub- scriber since such retransmission would mutilate the sig- nals. Accorditigly, a control circuit is provided for each sub-scriber transmission circuit to block the sending leg 50 when signals are received by the associated receiving leg. Other subs6ribers, however, must be permitted to inter- rupt the sending subscriber by transmitting into the hub predetermiiied signals such as a spacing "break" signal. In prior arrangements, this function is provided by a 55 doixble space detector individual to each subscriber which detects wheii: the individual subscriber and another sub- scriber simultaneously send spacing signals into the hub and, in response thereto, unblocks the sending leg. Where the hub serves a large number of subscribers, however, the 60 provision of doilble space detectors for each subscriber is relatively expensive. In addition, when two subscribers are simultaneously sending data signals into the hu , gar'b led data may be trangmitted to the other subscribers who are unaware that this is caused by a subscriber break- 65 ing in rather than by line hits, for example. Accordingly, it is an object of this invention to pro- vide a simple and economical control circuit for a hub concentration. sending leg of the sendin subscriber is unblocked by the I 9 double space detector, the sending subscriber is also advised of the interruption. The foregoing and other objects and features of this invention will be fully understood from the following d escription of an illustrative embodiment thereof taken in conjunction with the accompanying drawing which shows, in schematic form, the details of a hub control circuit for a hub repeater system in accordance with this invention. Referring now to the drawing, the hub circuit shown therein includes receiving hub RH and sendi'ng hub SH. Receiving hub R-H receives signals by way of lead R-1 !rom a receive leg, generally indicated by block 101, and is further multipled to other similar receiving legs via leads R-2 through R-n. The hilb voltage at terminal RH is provided by way of ground through breakdown diodes D4 and D5 and to negative battery by way of resistor 116. This maintains receiving hub RH during normal idle or marking conditions at, for example 6 volts nega- tive with respect to ground by virtue of the voltage drop across diode D4. When incoiriing signals are received by receiving hub RH from'a receiving leg, these signals are repeated by regenerative repeater 105 to sending hub SH or, in the alternative, directly applied to sending hub SH by way of lead 106. Sending hub SH, in turn, applies the signals in parallel to leads S-1 through S-n to the send legs such as the send leg connected to lead S-1 and generally indicated by block 102. It is noted that receive leg 101 receives signals via incoming line 103 and send leg 102 sends signals over.outgoing line 104. Lines 103 and 104, in tum, comprise a subscriber channel arranged, in accordance with this embodiment, for half-duplex operation. Signais received by receive leg 101 by way of incoming line 103 constitute negative signals when the line is idle or, alternatively, negative marking and positive spacing signals when inconiing data signals are being received. The application of the negative marking signal to the base of transistor Q1 -maintains the transistor nonconductive, permitting the application of positive battery to its

collector. This positive collector potential is applied to the base of transistor Q2 and the latter transistor is also maintained nonconductive. Accordingly; during the reception of marking signals no current flows into the receiving hub which is permitted to retain the negative hub voltage, as previously described. In the event that a.@positive spacing signal is received over line 103 and thus applied to transistor Ql, the transistor turns ON, bringing the collector voltage down to ground. This ground potential is applied to the base of transistor Q2 turning it ON, whereby emitter-to-collector current is provided to receiving hub RH. This emitterto-collector current is derived from positive battery by way of breakdown diode D-2 thus applying to receiving hub RH a positive voltage with respect to ground. Accordingly, the potential on receiving hub RH is negative when marking or idle signals are being received and goes positive when a spacing signal is received by a receiving It is another object of this invention to preclude garbied 7(leg such as receive leg 101. transmission when the sending subscriber is interrupted As previously disclosed, signals on receiving hub RH by a second subscriber. are repeated to sending hub SH and thence to the send

3;443,022 3 legs. The application of the negative marking signal to send leg 102 by way of lead S-1 lowers the emitter potential of transistor Q3 whereby the transistor turns ON. This applies a ground potential to the base of transistor Q4 and the latter transistor thus cannot conduct. Accordingly, positive battery is applied by way of resistor 120 to the base of transistor Q6, turning OFF this transistor whereby a negative potential is applied to lead 104 by way of resistor 121. Lead 104, in turn, preferably extends to the sending modulator or the outgoing line. Thus, when a negative marking signal is received by sending hub SH and applied to a sending leg such as send leg 102, a negative potential corresponding to a marking signal is, in turn, applied to the outgoing line such as lead 104. In the event that receiving hub RH has applied thereto a positive spacing signal, this positive signal is repeated to sending hub SH and thence to send leg 102, for example, by way of lead S-1. The consequent application of a positive potential to the emitter of transistor Q3 turns the transistor OF'F. Positive battery is thus applied to the base of transistor Q4. As described hereinafter, if the signals are not being received by the receive leg associated with the same subscriber as the send leg, the emitter of transistor Q4 has a potential close to ground applied thereto. Consequently, the application of positive battery to the base of transistor Q4 turns the transistor ON, thereby applying this emitter ground potential by way of its collector to the base of transistor Q6. Transistor Q6 thus turns ON and the positive potential applied to its emitter drives its collector positive. Thus, the application of a positive spacing signal to send leg 102 by send hub SH results in the application of a positive spacing signal to outgoing line 104. The receive leg and the send Ieg of each subscriber is arranged to preclude the reception of signals from the hub when the subscriber is sending signals into the hub. This is accomplished by disabling transistor Q4 when signals are being received by the receive leg. Assuming now that an idle or marking signal is applied to transistor Ql by way of lead 103, transistor Ql is turned OFF, as previously described, rendering its collector positive. This positive potential is applied through breakdown diode Dl to lead 117. Diode Dll in send leg 102, however, precludes the application of the positive potential on lead 117 therethrough to capacitor 112. Accordingly, capacitor 112 is charged to positive battery by way of resistor 123. This positive charge, in turn, is applied to the base of transistor Q5 turning it ON. Thus the collector of transistor Q5 is brought down to ground potential and this ground potential is applied to the reversely poled diodes 114. Since the breakdown voltage of diodes 114 is small, the potential applied to the emitter of transistor Q4 is only slightly positive with respect to ground. With the potential on the emitter of transistor Q4 brought down to this slightly positive voltage, the transistor is enabled to pass spacing signals therethrough, as previously described. Recalling now that the application of a spacing signal to transistor Ql turns the transistor ON thereby bringirig its collector potential down to ground, it is noted that this ground potential is also applied through breakdown diode DI. This drives the potential of lead 117 negative by virtue of the connection of negative battery thereto by way of resistor 118. The negative potential is then applied through diode Dll of send leg 102 to capacitor .112. Since the negative charge on capacitor 112 is also applied to the base of transistor Q5, the latter transistor is turned OFF. This removes the ground applied to the emitter of

transistor Q4 and positive battery is thus provided to the emitter. Accordingly, transistor Q4 does not turn ON when transistor Q3 applies a positive signal to the base of transistor Q4 in response to the reception of a spacing signal from sending hub SH. Thus, when spacing signals are received by a receive leg, transistor 4 Q4 of the associated send leg is maintained OFF to preciude the repeating of the signals to the sending subscriber. When the spacing signal from line 103 terminates, the negative potential applied to lead 117 is removed and capacitor 112 is again charged via resistor 123 to turn ON transistor Q5 and re-enable transistor Q4. It is noted, wever, that capacitor 112 provides a delay in the returning of transistor Q5 to the ON condition. This 10 is to compensate for the delay of regenerative repeater 105 in repeating the signals to send leg 102 via send hub SH. Receiving hub RH is also connected to a double space detector and break transmitter, generally indicated by 15 block 110. The function of the double space detector and break transmitter is to determine when a second subscriber breaks into the transmission of a first subscriber and upon the determination thereof to indicate to all subscribers, including the original sending subscriber, that a 20 second subscriber has transmitted a space or break or attempted to interrupt the transmission of the sending subscriber. Detecti<) n of a double space, that is, the transmision of spacing signals by two or more subscribers into receiv- 25 ing hub RH, is provided by transistor Q12. The base of transistor Q12 is connected to negative battery by way of breakdown diode D7. The emitter of transistor Q12 extends by way of breakdown diode D6 to receiving bub RH. It is recalled that when all receiving le-s are sending 30 idle or marking signals into receiving hub RH, no current flows thereto and receiving hub RH has a potential negative with respect to ground. This potential is insufficient to break down diode D6 and thus the emitter potential is the same as the base and transistor Q12 does not conduct. 35 Accordingly, the collector of transistor Q12 has a negative potential applied thereto by way of resistor 125. When a receiving leg, such as receiving leg 101, receives a spacing signal, transistor Q2 supplies current to receiving hub RH and the hub potential is raised above 40 ground, as previously described. This potential is just sufficient to break down diode D6. Virtually all of the current, however, from receive leg 101, passes through resistor 116 to negative battery. Accordingly, very little current is applied to the collector or transistor Q12 from 45 the emitter. Consequently, there is virtually no current flow through resistor 125 to negative battery and the coliector of transistor Q12 is- maintained negative at substantially the same voltage level as previously described with respect to the reception of idle or marking signals by 50 receiving hub RH. Assuming now that a second subscriber sends a spacing or break signal, additional current now flows from the silbscriber receiving leg into receiving hub RH. Since the spacing signal from the first subscriber breaks down 55 diode D6, as previously described, the additional spacing current from the second subscriber is permitted to pass through diode D6, the emitter-to-collector path of transistor Q12 and resistor 125 to negative battery, substantially raising the potential of the collector of tran- 60 sistor Q12 toward ground. Thus, it is seen that the collector of transistor 4QI2 is maintained negative unless two or more subscribers simultaneously transmit spacing signals into receiving hub RH whereupon the collector potential is driven positive toward ground. The break transmitter portion of the circuit includes transistors Q8 and Q9, which transistors are arranged as a monostable multivibrator. In the normal condition, with the base of transistor Q9 connected to positive battery by way of resistor 129, this latter transistor is 70 maintained ON. This brings the collector voltage of- transistor Q9 down t'oward ground, and this ground potential is applied to the base of transistor Q8 by way of resistor 131. Since, at the same time, the base of transistor Q8 is connected to the negative potential at the collector 75 of transistor Q12 by way of lead 126, the, rumulative

82443,022 5 effect is to maintain the base of transistor QS below groun d, thereby maintaining the latter transistor OFF. Assu ming now that a double space is received by receiv- ing hub RH, the collector of transistor Q12 is driven posi- tive toward ground, as previously described. Accordingly, 5 the potential on the base 6f transistor Q8 is corresponding- ly raised until it is brought above ground by the positive batter y connected thereto by way of resistor 127. Ac- cordin gly, transistor Q8 turns ON and the resultant col- lector- to-emitter cuffent removes the positive potential 10 applie d to the collector by way of resistor 133. This neg- ative- going transistion at the collector of transistor Q8 is passe d through

capacitor 128 to the base of transistor Q9 turning the latter transistor OFF. The collector of transis tor Q9 is thus driven positive, feeding back a po- 15 sitive-going potential to the base of transistor Q8 by way of resistor 131 and shunting capacitor 130. At this time, capaci tor 128 again begins to charge by way of resistor 129 to positive battery. After a predetermined interval of time corresponding to a break signal interval, the 20 charg e on capacitor 128 is raised sufficiently to turn tran- tor Q9 back ON. This results in a negative going tran- sition at the collector of transistor Q9, which transition is passed through capacitor 130 to the base of transistor Q8 turning the lafter transistor OFF. Accordingly, the 25 multivi brator restores to its initial condition. When the multivibrator is at its normal quiescent con- dition, transistor Qg is turned OFF, as previously de- scribe d, and its positive collector potential is applied to the base of transistor Q7. This maintains transistor Q7 30 OFF and precludes the passage, of emitter-t-o-collector curre nt therethrough. When the multivibrator is driven to the off-normal condition in response to the reception of a double space, transistor Q8 turns ON lowering the base potent ial on transistor Q7. This permits emitter-to-col- 35 lector current flow and with the emitter of transistor Q7 conne cted to positive battery by way of breakdown diode D9 and the collector of transistor Q7 connected to receiv- ing hub RH, a simulated spacing signal is thus transmitted 40 into the hub. Since the multivibrator remains off-normal for an interval corresponding to the duration of a break signal, ia simulated break signal is thus transmitted into a hub when a double space is detected. As previously described, in the quiescent condition of the multivibrator, transistor Q9 is conducting. Accord- 4r, ingly, its collector-to-emitter current is applied to the base of transistor QIO and the latter transistor is also con- ductin g. This brings the collector potential of transi@stor Q10 down to ground. 50 The collector of transistor QIO is connected by way of breakdown diode DS to -the base of transistor Qll. Since, as previously described, the collector -potential of transistor QIO is eIGse to ground, the drop across diode D:8 renders the base of transistor Qll negative r)5 with respect to ground. Since transistor Ql:1 is connected as an emitter follower, the emitter is coffespondingly negati ve with respect to ground, thereby applying a nega- tive potential to lead 135. Lead 135, in turn, is multi- plied to leads DC-1 through DC-21 which latter leads 60 then extend to corresponding ones of the send legs. Re- ferrin g now to send leg 102, it is noted that lead 135 is connected to diode DIO which, in turn, is connected to capacitor 112. Since, in the quiescent condition Of the multivibrator, lead 135 has a negative potential ap- plied thereto, diode D10 functions to block this poten- 65 tial and consequently disconnecting lead 135 from capaci- tor 112. Similarly, corresponding diodes such as diode DIO in each of the other send legs, function to decouple lead 135 from the associated delay capacitors 112 in each of the send legs. 70 Assu ming now that a d-ouble space is detee-ted and t@he multivibrator is driven to the off-normal condition, transis tor Q9 is turned OF'F, as previously described. T-his stops the current flow into the base of transistor QIO and 75 6 the latter transistor turns OFF. With transistor QIO turned OFF, positive battery is applied to the collector thereof by way of resistor 136. This increased positive potential thus raises the potential on the base of transistor Qll to above ground. The emitter of transistor Qll following the base potential is also raised above ground. Accordingly, lead 13,5 is driven positive and this positive potential is applied by way of leads DC-1 through DC-n, diodes DIO in each of the send legs to the associated capacitors 112. Assuming now that the subscriber associated with lines 103 and 104 is sending when the double space is detected, it is recalled that the reception of spacing signals by receive leg 101 provides a negative p(>tential by way of lead 117 and diode DII to capacitor 112. This, in tum, turns OFF transistor Q5 whereby transistor Q4 is disabled to preclude the repeating of the signals back to the subscriber, as previously described. When the double space is detected, however, lead 135 is driven positive, as previously described, and this positive potential is provided by way of diode DIO to t@he base of transistor Q5. Accordingly, transistor Q5 is forced to turn ON, enabling transistor Q4. Since, as previously described, transistor Q7 is sending a spacing break signal into receive hub RH, with transist6r Q4 enabled, the spacing break signal is repeated through sending hub SH, send leg 102 and line 104 back to the subscriber even though he is presently sending. Thus, it is seen that t d bling feature in send leg 102 is removed in the event that a double space is detected and a break signal is returned to alI including the sending subscriber. What is claimed is:

1. @In a hub type data repeater system wherein a plurality of data channels are

interconnected by way of a common hub, a receiving leg individual to each channel for applying data signals received f@r6in said channel to said common hub, a sending leg individual to each channel for repeating said data signals applied to said common hub to said channel, a control circuit individual to each channel and responsive to data signals received from said channel for blocking said sending leg individual thereto, and means common t-o said plurality of channels and responsive to the simultaneous reception of signals from two or more channels for precluding the operation of all of said control circuits. 2. In a hub type data repeater system in accordance with claim 1 wherein said common means includes means for applying a predetermined signal to said hub when said operations of said control circuits are precluded. ' 3. In a hub type data repeater system wherein a plurality of data channels are interconnected by way of a common hub, a receiving leg individual to each channel for applying data signals received from said channel to said common hub, a sending leg individual to each channel for repeating said data signals applied to said comMon hub to said chan'nel, and means common to said plurality of channels and responsive to the simultaneous reception of signals froin two:or -more channels for sending a predetermined sigfial to all of said ch'annels. 4. In a hub type data repeater system wherein a plurality of data channeis are interconnected by way of a common hub, a receiving leg individual to each channel for applying data signals received from said channel to said common hub, a sending leg individual to each channel for repeating said data signals applied to said common hub to said channel, a control circuit individual to each channel and responsive to data signals received from said channel for blocking said sending leg individual thereto, and means egmmon to sa:id plurality of channels and responsive to the simultaneous reception of signals from two or more channels for sending a predetermined signal to all of said channels. 5. A data signal repeater system comprising a plurality of data channels, a common hub, a receiving leg associated with each channel for -applying signals received from

3)443)022 7 said channel to said hub, a sending leg associated with each channel for sending said signals applied to said hub to said channel, signal detector means connected to said hub for detecting the simultaneous application of signals to said hub by two of said receiving legs, and means responsive to said signal detector means for applying a predetermined signal to all of said channels. 6. A data signal repeater system comprising a plurality of data channels, a common hub, a receiving leg associated with each channel for applying signals received from said channel to said hub, a sending leg associated with each channel for sending said signals applied to said hub to said channel, control means individual to each channel and responsive to signals received from said channel for blocking said sending leg associated thereto, signal detector means connected to said hub for detecting the simultaneous application of signals to said hub by two of said receiving legs, and means responsive to said signal detector means for applying a- predetermined signal to all of said channels. 7. A data signal repeater system comprising a plurality of data channels, a common hub, a receiving leg associated with each channel for applying signals received from said channel to said hub, a sending leg associated with each channel for sending said signals applied tO said hub to said channel, control means individual to each channel and responsive to signals received from said channel for blocking said sending leg associated thereto, signal detector means connected to said hub for 8 hub by two of said receiving legs, and means responsive to said signal detector means for disabling all of said control means. 8. A data signal repeater system comprising a plurality of data channels, a common hub, a receiving leg associated with each channel for applying signals received from said channel to said hub, a sending leg associated with each channel for sending said signals applied to said hub to said channel, control means individual to each 10 channel and responsive to signals received from said channel for blocking said sending leg associated thereto, signal detector means connected to said hub for detecting the simultaneous application of signals to said hub by two of said receiving legs, means responsive to said signal 15 detector means for disabling all of said control means, and other means responsive to said signal detector means for applying a predetermined signal to said hub. References Cited 20 UNITED STATES PATENTS 2,542,208 2/1951 Purris ---------- 178-2 2,607,852 8/1952 Rea ---------- 178-2 2,639,320 511953 Gardner ---------- 178-2 25 2,994,736 8/1961 Hopner ---------- 178-2 THOMAS B. HABECKER, PrimaryExamitier. U.S. Cl. X.R. detecting the simultaneous application of signals to said .1(307-231

☐ 47. Document ID: US 2877456 A

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US-PAT-NO: 2877456
DOCUMENT-IDENTIFIER: US 2877456 A

TITLE: Zero speed detector

DATE-ISSUED: March 10, 1959

US-CL-CURRENT: 361/22; 324/161, 324/772, 340/671

DOCUMENT TEXT:

March 10, 1959 S. BENOWITZ 2,877,456 ZERO SPEED DETECTOR Filed S6pt. 26, 1956 2 Sheets-Sheet I /7 16 z Z7 V 14 V 15 v/6 86.6 .3 0 6 Z70' c@ gg INVENTOR. .9 W17-Z B Y -Alr

March 10, 1959 Filed Sept. 26, 1956 S. 13ENOWITZ ZERO SPEED DETECTOR *o/ 4f, 4.9 45 4 .@z BY 2 877,456 2 Sheets-Sheet 2 4 3 INVENTOR. d 19 7 7',02@;Awll6c- oPi@T e-IV 7-

United States Patent Office 21877@456 2,877,456 ZF,RO SPEED DETE4CTOR 5 Sander Benowitz, Mountain View, Calif., assignor, by mesne assignments, to the United States of America as represented by the Secretary of the Air Force Application September 26, 1956, Serial No. 612,344 1 10 3 CWms. (C]. 340-271) This invention relates to a zero speed detector to deter- 15 mine whether an object, such as a rotating shaft, has ceased all motion and is at rest. Another object is a provide a device which will give an indication at a point remote from an object, such as a shaft, whether said object has ceas@-d all motion 20 and is at rest. Another object is to provide a device whicii will deter- mine when the speed, of a compressor shaft for a wind tunnel is at zero or near zero. A further object is to provide a device that will per- 25 for a definite function, stich as controlling the coupling or uncoupling of a compressor for a wind tunnel. A still further object is to provide a device to deter- mine whether an object, which may have either rotary or straight line motion, has reached zero speed or is 30 near zero speed. These above objects are accomplished by providin.- a sensing device and a measuring element. The sensing device may be either a standard synchro-tie transmitter, which is driven by the shaft or a plurality of differential transformers, while the measuring element may consist of either a plurality of indicating lamps or a plurality of standard over-voltage relays. Wben used with a compressor shaft for a wind tunnel the indicating elements may be located on an operator's 4 0 panel at a place remote from the compressor. The standard synchro-tie transmitter has a rotor with one winding which is excited witli single phase alternating ctirrent and three stator windings which are spaced 120 @clegrees apart and brolight out to thre-- - terminals. When 45 at standstill, single phase from th.- rotor is induced by transformer action into the stator windings. Since the rotor can have any position the stator winding voltages will depend on the relative position of the rotor with respect to the stator. A simple indicator consists of three -50 indicating lamps coilnected to the terminals of the syn- chro-tie transmitter. The three Jamps will be lighted to varying de.-rees of illumination depeiiding upon the position of the rotor with respect to the stator windings. As the rotor moves the lamps will change in intensity of 55 illumination and a ebange in intensily of the light will indicate rotation of the shaft whereas when the change in intensity stops, zero speed for the shaft is indicated. By observing the relative intensity of the lanips it is possible to detect Yery minute changes in position of the shaft. 00 Lamp systems provide only a visual indication for zero speed and therefore are not satisfactory for all applica- tions. When a measuring device is

required that will perform a definite function, such as controlling the cou- pling or uncoupling a compressor for a wind tunnel, over- 65 voltage relays are connected across the three synchro-tie transmitter terminals. These are standard relays which close their contacts at or above a definite voltage setting. The relays can be set to close their contacts within a definite time delay period. Therefore the relays cart be 70 set so that the shaft must move at a very slow speed befote any of the i-elays can close their contacts. Patented Mar. 10, 1959 2 While the device using the synchro-tie transmitter is -tpplicable in many cases it is not suitable where temperatures in the order of 600' F. are anticipated nor where the motion being check-ed is straigbt line motion. A device which is capable of more general use is a systern which uses a plurality of differential- transformers. In the case where the object is a shaft, a disk with a pliirality of projections of magnetic material is mounted on the end of the shaft. The differential transformers are mounted adjacent the disk. The differential transformers have a primary winding on one leg and two secondary Nvindings, wound in opposition on two other legs. A fixed element of magnetic material is located adjacent the transformer core so as to provide a constant length air gap in the ma.-netic circuit through one of the secondary windings. The projections on the disk acts as a means to provide a variable gap length in the magnetic circuit, through the other of the secondary windings. As the disk rotates the projections will enter and leave the ma.netic path causin.- an unbalance in the magnetic circuits, thereby causing a difference in the induced voltage in the two secondary windings. By proper shaping of the air gap the envelope of the induced voltage can be made to follow a sine wave. By spacing the differential transformers 120 electrical degrees apart the envelopes for the volta@es will be similar to those for the synchro-tie system. The measurin.- elements may therefore be tbisame as for the synchro-tie transmitter system. In the drawings: Figure I is a schematic wirin.- diagram illustrating the arrangement and connection of the various elements in one embodiment of the invention. Figure 2 is a schematic wiring diagram of another enibodiment wherein over-volta.-e relays have been substituted for the indicating lamps, Figure 3 shows the ci-irves for the envelopes of the voltages indticed in the tbree stator windin.-s as the rotor moves throtigh various positions. Figure 4 shows a schematic diagram of aiiother embodiment wherein differential transformers are used as the sensing device. Referring more partic@alarly to Figure I the reference numeral 11 refers to a shaft whose speed is being measured. A rotor winding 12 is coupled to the shaft 11 either directly or throu,-h reduction .-earing, which will be explained in greater detail in connection with Figure 2. An afternatin.- cur.-ent source 13 is connected to the rotor winding 12. Three stator windings 14, 15 and 16 are spaced 120 degrees apart arotind th.- rotor ivindin.and are located in electromagnetic energy coupling relation with said rotor windings. Three indicating lamps 17, 18 and 19 are connected across the stator windings 14, 15 a-nd 16 respectively. In the operation of the device of Figure 1 the rotor winding 12 is driven by the shaft it, for which a zero speed indication is desired. The rotor is energized by an altemating current source 13. As the rotor winding is rotated througli various positions the induced voltages in the three stator windings follow the clirves as shown in Figtire 3. These voltages are impressed across the lamps 17, 18 and 19. Since the induced voltages in windings 14, 15 and 16 will change as the rotor winding rotates t'iie intensity of the illumination of the lamps vlill vary. Since the huinan eye will detect vel-y small changes in intensity of illumination, particularly if clear bulb type of incaiidc-,cent lanips are Lis--d, it is pos.,,ibl,-- to detect very minute changes in position or creepin.- of the shaft. The device of Figure 2 is similar to the device of Figure I but has three over-voltage relays 27, 28 and 29 substituled for i'ae iiidicaiing laiiips 1-i, 18 and 19. Like elements have been given the same reference numerals in both figures. In Fi-,Ure 2 the rotor winding 12 is

2,877,456 3 show.n as coupled to the - shaf, 11 throu,ah a reduction gearing system 29 and 21 and a second shaft 22. If the accuracy is not great enou.-h with the rotor windin- coupled directly to the, shaft as in Figure 1, the accuracy caii be increased eiiher by using a multi-pole synch@-c- tic 5 transmitter or by the use ofreduction gearing as sh()vin, The circuits controlled@by, the time delay relays 27 28 and 29 may be used to control the connection of coii.@linr number 52 to thereby control the operation o'L compre@ sor number 53. 10 The geannng system can be friction gearing or other knowntypeofgearing. The@rellayscanbesetsothattheir con'Lacts close at a voltage

Nvhich is slightly below 86.6% of the -maxir@ium stator terniinal voltages. Then a Telay ,;h will be.@closed fdr the portion of @the etii-ves , o-tvli by 15 the. shaded portion in Figure 3. F@gure 3 shows - that the shaded portions of the curves overlap for all values of 0. Therefore if 0 -has a fixed value corresponding to - t'ac position where -the Totor stops,,there will alvjays bc a cor- respoTid-'Ln.- shaded are,,i for whieti-one of t@ie threc@relays 20 will close. Since the @closing tin-ie of the relays can be made adji,@stable it can be made sufficiently long so that none of the relays w-ill close until the shaft reaches a very slow speed. In the explanation of the operation of the device (f 25 Fi,-iure 2, it will be assunied that the rotor is stopped at 0 equal to 30 degrees -,vith the voltages iiiduced in LTiz@ stator windin,-s being as sho-,vn in Figure 3 for this value of 0. It can be se--n tqen that relay 28 will be ener-ized. As the rotor starts to turn in a cuanter-clockwise direction 30 the voltage will decrease in winding 15 and increase in winding 14 until 0 equals 60 degrees at which time relay 28 will open its contacts and relay Z7 will start to clos-- its contact& If the speed of the shaft is great enough by this t:lme the rotor will turn to the point where 0 35 is equal to 120 degrees before the;contacts for relay 27 can become completely closed. Thereafter none of the relays will close their contacts until the speed of the shaft is auain reduced to the point where the time taken for the Totor to rotate tlroti.-h 60 degrees is greater than 40 the closing time for the relays. Though the red@.jction gearing is shown in connection with the device of Figiire 2 it is obviotis that it colld also be used with the device of Fi.-Ure 1. It is also obvious that the indicating lamps and relays could be 45 tised at the same time. The device of Figure 4 uses a plurality of differential transformers as the sensing element. These can be used for indicating the @speed of an object which has @either straight line motion or rotary motioii. In the device 50 of this figure they indicate the speed ot an object which has rotary motioii, such as a sha'tt 11. A disk 42, havin,- a plurality of prp:iections 43,of niagnetic@ material thereon is mounted on the end of the shaft 11. A plurality oi differential transformers 40, 40' Fnd 40" are located ad- 5-3 j,icent the disk. S,ationary armatures 44, 44' and 44" are located adjacent but spaced from said disk, Cores 45. 45' and 45" of said transformers qre lo-.ated between sai'd stationary armatures and said disk so as to form air ,g4r,s 46, 46' and 46" between ttie cores and the stationarv armatures and gkns 47, 47' and 47" betweeja the cores a Ti Cl the disk, Cores 45, 45' and 45" have primary windin- ,s 48@ 43' and 48" on on--le-. and secondary wind 49, 50, 49', 50' and 49", 50" resp-.ctively wound ti@,o other legs. The windings 49, 49' and 49" are wound c@ in cpposition to thewindings 50, 50', 50". 'nie outputs of the s-.candaries are connected to relays 51, 51' and 51". It is obvious that indicating lamps such as those of Figure I could also be used. In th.-operation of the device of Figure 4 ;.t can.be 70 seen the,, since armature 44 is stationaly and core 45 is stationary the air gap 46 will remain constant. It can also be seen that the air -ap 47 will change as the disk 4 42 rotates., @Vith Lhe projectioii-43. loqate@d -ad jacemt..trans:. former 40 as shown the air gaps in the two magnetic circiiits through the two secondary windings 49 and 50 Nvill be equal and the output voltage arross the two secondary windings will be zero. As the disk rotates the projections will ent-.r and leave the magnetic path, thereby changing the air gip leii-th -.'n one of t'ile niagnetic circuits of the transformer so that the voltages induced in the tv@o secondaries not cancel ca-@h other. With lircper shaping of the air - aps the envelope of the volta.@ecanbe-madetofollowasinewave.. Vlithtransformers 40, 40' and 40" spaced 120 electrical degrees apait the envelopes for the voltages will be similar.to those shown in Figure 3. The measuring.clement wfll operate in the ame inan@,-r as e,-@plain-,d in the operation of the devices s of- Figures I and 2. Tlere is thus providcj ,in acrurate device for indicating ivhcn,,the:speed of an object, such as:a rotating shaft, decreases, to..,zero.or very near zero. While certain embodinierts of tLe invvntioja have been desqribed in ,;ome detail, it v.,ill be iinderstood that numerous changes may be -iiade without departing from the general pr- @Dciples ,lnd scope of the invention.)Vhat is clain-led is: 1. A device responsive to the inotion of an object,comprising; a pl,.irality of diterential transformers located adjacent said object and each having a primary vinding and two,secondary windilngs wound in opposition thereon, an alternatir.,g current source connected to said primary, windings, mearis coupled. to said object for v@rying the cotipling between the primary winding and one of said secondary wi-tid:.ngs ei. each transformer to generate a plurality of altarnat;nl- voltages having sinusoidally,:var-ying envelopes of equal amplitudes with periods ivbich,are equ-,ii

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☐ 1. Document ID: US 20040014710 A1

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TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

PUBLICATION-DATE: January 22, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/45; 514/263.37

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NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton	MA	US	

US-CL-CURRENT: 514/12

ABSTRACT:

The present invention provides a method for treating and/or preventing damage to a retina or optic nerve in a subject comprising administering to the subject a therapeutically effective amount of oncomodulin. Preferably, the subject is a mammal, most preferably, a human. In preferred embodiments, the oncomodulin may be used in combination with mannose, a mannose derivative and/or inosine.

☐ 3. Document ID: US 20020160933 A1

L4: Entry 3 of 15 File: PGPB Oct 31, 2002

PGPUB-DOCUMENT-NUMBER: 20020160933
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020160933 A1

TITLE: Methods and compositions for producing a neurosalutary effect in a subject

PUBLICATION-DATE: October 31, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/1

ABSTRACT:

Methods and compositions for producing a neurosalutary effect in a subject are provided. These methods generally involve administering to a subject a therapeutically effective amount of a compound that modulates the activity of N-kinase, or analog thereof. Pharmaceutical and packaged formulations including the compounds of the invention, e.g., compounds that modulate the activity of N-kinase, are also provided.

☐ 4. Document ID: US 20020137721 A1

L4: Entry 4 of 15 File: PGPB Sep 26, 2002

PGPUB-DOCUMENT-NUMBER: 20020137721
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020137721 A1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

PUBLICATION-DATE: September 26, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/45

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Des

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☐ 5. Document ID: US 20020128223 A1

L4: Entry 5 of 15 File: PGPB Sep 12, 2002

PGPUB-DOCUMENT-NUMBER: 20020128223
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020128223 A1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

PUBLICATION-DATE: September 12, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/45

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Des

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☐ 6. Document ID: US 20020119923 A1

L4: Entry 6 of 15 File: PGPB Aug 29, 2002

PGPUB-DOCUMENT-NUMBER: 20020119923
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020119923 A1

TITLE: Methods and compositions for producing a neurosalutary effect in a subject

PUBLICATION-DATE: August 29, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Square	MA	US	

US-CL-CURRENT: 514/12; 514/47, 514/729

ABSTRACT:

Methods and compositions for producing a neurosalutary effect in a subject, such as modulating neuronal survival and/or regeneration in a subject, are provided. Pharmaceutical and packaged formulations are also provided.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Des

☐ 7. Document ID: US 20020055484 A1

L4: Entry 7 of 15 File: PGPB May 9, 2002

PGPUB-DOCUMENT-NUMBER: 20020055484
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020055484 A1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

PUBLICATION-DATE: May 9, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/45

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Des

☐ 8. Document ID: US 20020042390 A1

L4: Entry 8 of 15 File: PGPB Apr 11, 2002

PGPUB-DOCUMENT-NUMBER: 20020042390

PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020042390 A1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

PUBLICATION-DATE: April 11, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Benowitz, Larry I.	Newton Centre	MA	US	

US-CL-CURRENT: 514/45; 514/263.37

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

☐ 9. Document ID: US 6551612 B2

L4: Entry 9 of 15 File: USPT Apr 22, 2003

US-PAT-NO: 6551612
DOCUMENT-IDENTIFIER: US 6551612 B2

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

DATE-ISSUED: April 22, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Benowitz; Larry I.	Newton Centre	MA		

US-CL-CURRENT: 424/450; 424/422, 424/423, 424/484, 424/486, 424/489, 424/490, 424/497, 514/45

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

11 Claims, 16 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 9

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Des

☐ 10. Document ID: US 6440455 B1

L4: Entry 10 of 15 File: USPT Aug 27, 2002

US-PAT-NO: 6440455
DOCUMENT-IDENTIFIER: US 6440455 B1

TITLE: Methods for modulating the axonal outgrowth of central nervous system neurons

DATE-ISSUED: August 27, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Benowitz; Larry I.	Newton Centre	MA		

US-CL-CURRENT: 424/450; 424/422, 424/423, 424/484, 424/486, 424/489, 424/490, 424/497, 514/45

ABSTRACT:

Methods for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons are also provided. Finally, a packed formulation comprising a pharmaceutical composition comprising an inosine nucleoside and a pharmaceutically acceptable carrier packed with instructions for use of the pharmaceutical composition for treatment of a central nervous system disorder is provided.

11 Claims, 16 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 9

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Des

☐ 11. Document ID: US 5898066 A

L4: Entry 11 of 15 File: USPT Apr 27, 1999

US-PAT-NO: 5898066
DOCUMENT-IDENTIFIER: US 5898066 A

TITLE: Trophic factors for central nervous system regeneration

DATE-ISSUED: April 27, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Benowitz; Larry I.	Newton	MA		
Irwin; Carleen A.	Newton	MA		
Jackson; Paul	Brookline	MA		

US-CL-CURRENT: 530/300; 530/399

ABSTRACT:

Cell culture conditions were developed which maintain the nerve cells of the retina in well-defined, serum-free conditions. The molecular factors that stimulate axonal regeneration from these neurons were characterized. The glial sheath cells that surround the axons of the optic nerve release two molecules that trigger and sustain nerve regeneration. One of the molecules is referred to as axogenesis factor 1 (AF-1), and is a low molecular weight polypeptide with a size in the range of 1000 daltons. The second molecule, AF-2, is a larger protein with a size of approximately 12,000 daltons. Studies indicate that these factors are strongly involved in CNS regeneration, and are therefore useful in the treatment of spinal cord and other nervous tissue damage.

1 Claims, 12 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 5

☐ 12. Document ID: WO 2004028468 A2

L4: Entry 12 of 15 File: EPAB Apr 8, 2004

PUB-NO: WO2004028468A2
DOCUMENT-IDENTIFIER: WO 2004028468 A2
TITLE: METHODS AND COMPOSITIONS FOR TREATMENT OF NEUROLOGICAL DISORDER

PUBN-DATE: April 8, 2004

INVENTOR-INFORMATION:

NAME	COUNTRY
BENOWITZ, LARRY I	US

INT-CL (IPC): A61 K 0/

ABSTRACT:

CHG DATE=20040420 STATUS=O>The present invention provides methods and compositions for producing a neurosalutary effect in a subject useful in treatment of neurological disorders, including retinal and optic nerve damage, in a subject in need thereof. The method includes administering to a subject a therapeutically effective amount of a hexose, such as mannose.

☐ 13. Document ID: WO 9911274 A1

L4: Entry 13 of 15 File: EPAB Mar 11, 1999

PUB-NO: WO009911274A1
DOCUMENT-IDENTIFIER: WO 9911274 A1
TITLE: USE OF PURINE NUCLEOSIDES FOR MODULATING THE AXONAL OUTGROWTH OF CENTRAL NERVOUS SYSTEM NEURONS

PUBN-DATE: March 11, 1999

INVENTOR-INFORMATION:

NAME	COUNTRY
BENOWITZ, LARRY I	US

INT-CL (IPC): A61 K 31/70; A61 K 31/52
EUR-CL (EPC): A61K031/52; A61K031/70, A61K031/70

ABSTRACT:

CHG DATE=19990905 STATUS=O>Methods and compositions for modulating the axonal outgrowth of central nervous system neurons are provided. Methods for stimulating the axonal outgrowth of central nervous system neurons following an injury (e.g., stroke, Traumatic Brain Injury, cerebral aneurism, spinal cord injury and the like) and methods for inhibiting the axonal outgrowth of central nervous system neurons in conditions such as epilepsy, e.g., post-traumatic epilepsy, and neuropathic pain syndrome, are also provided. These methods generally involve contacting the central nervous system neurons with a purine nucleoside, or analog thereof. Preferably, inosine or guanosine is used to stimulate axonal outgrowth and 6-thioguanine is used to inhibit axonal outgrowth. The methods and compositions are particularly useful for modulating the axonal outgrowth of mammalian central nervous system neurons, such as mammalian retinal ganglion cells. Pharmaceutical and packaged formulations that include the purine nucleosides, and analogs thereof, of the invention are also provided.

☐ 14. Document ID: WO 9606859 A1

L4: Entry 14 of 15 File: EPAB Mar 7, 1996

PUB-NO: WO009606859A1
DOCUMENT-IDENTIFIER: WO 9606859 A1
TITLE: TROPHIC FACTORS FOR CENTRAL NERVOUS SYSTEM REGENERATION

PUBN-DATE: March 7, 1996

INVENTOR-INFORMATION:

NAME	COUNTRY
BENOWITZ, LARRY I	
IRWIN, CARLEEN A	
JACKSON, PAUL	

INT-CL (IPC): C07 K 14/475; A61 K 38/00; C07 K 16/22; C12 N 15/12
EUR-CL (EPC): C07K014/475

ABSTRACT:

Cell culture conditions were developed which maintain the nerve cells of the retina in well-defined, serum-free conditions. The molecular factors that stimulate axonal regeneration from these neurons were characterized. The glial sheath cells that surround the axons of the optic nerve release two molecules that trigger and sustain nerve regeneration. One of the molecules is referred to as axogenesis factor 1 (AF-1), and is a low molecular weight polypeptide with a size in the range of 1000 daltons, determined to be about 707 daltons by mass spectroscopy. The second molecule, AF-2, is a larger protein with a size of approximately 12,000 daltons. Studies indicate that these factors are strongly involved in CNS regeneration, and are therefore useful in the treatment of spinal cord and other nervous tissue damage.

☐ 15. Document ID: WO 9408618 A1

L4: Entry 15 of 15 File: EPAB Apr 28, 1994

PUB-NO: WO009408618A1
DOCUMENT-IDENTIFIER: WO 9408618 A1
TITLE: ORAL TOLERANCE AND IMMUNE SUPPRESSION IN THE TREATMENT OF AIDS

PUBN-DATE: April 28, 1994

INVENTOR-INFORMATION:

NAME	COUNTRY
BENOWITZ, LARRY I	
TRUJILLO, J ROBERTO	
IRWIN, CARLEEN A	

INT-CL (IPC): A61K 39/12; C12Q 1/68; C07K 3/00
EUR-CL (EPC): C07K014/47

ABSTRACT:

A method of diagnosis and treatment of AIDS-related disorders has been developed based on the presence of an autoimmune response, evoked by infection with the human immunodeficiency virus (HIV), which then leads to the destruction of cells in the immune and nervous systems.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc

Terms	Documents
Benowitz-Larry-I.IN.	15

Hit List

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Search Results - Record(s) 1 through 9 of 9 returned.

☐ 1. Document ID: AU 2003272728 A1, WO 2004028468 A2

Using default format because multiple data bases are involved.

L5: Entry 1 of 9 File: DWPI Apr 19, 2004

DERWENT-ACC-NO: 2004-316013
DERWENT-WEEK: 200462
COPYRIGHT 2004 DERWENT INFORMATION LTD
TITLE: Use of hexose (e.g. D-mannose) to treat/alleviate neurological disorders such as traumatic brain injury, stroke, cerebral aneurysm, Parkinson's disease, amyotrophic lateral sclerosis and Alzheimer's disease

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2002US-414063P (September 27, 2002)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 2003272728 A1	April 19, 2004		000	A61K000/00
WO 2004028468 A2	April 8, 2004	E	059	A61K000/00

INT-CL (IPC): A61 K 0/00

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc

☐ 2. Document ID: US 20030153501 A1

L5: Entry 2 of 9 File: DWPI Aug 14, 2003

DERWENT-ACC-NO: 2003-787289
DERWENT-WEEK: 200374
COPYRIGHT 2004 DERWENT INFORMATION LTD

TITLE: Treatment and/or prevention of ocular disorders, e.g. retina or optic nerve damage, comprises administering oncomodulin

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2002US-0294965 (November 14, 2002), 2001US-0872347 (June 1, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 20030153501 A1	August 14, 2003		006	A61K038/17

INT-CL (IPC): A61 K 38/17

ABSTRACTED-PUB-NO: US20030153501A
BASIC-ABSTRACT:

NOVELTY - Ocular disorders are treated and/or prevented by administering oncomodulin.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for an article of manufacture comprising packaging material with a label indicating administration, and oncomodulin.

ACTIVITY - Ophthalmological. No biological data given.

MECHANISM OF ACTION - Adenylate cyclase activator; Macrophage activator; Phosphodiesterase (IV) inhibitor; beta -2 adrenoreceptor inhibitor; beta -2 adrenoreceptor agonist. No biological data given.

USE - The method is used for treating and/or preventing damage to a retina or optic nerve, including damage from ischemic or hypoxic stress, excess intraocular pressure, or injury, in a mammal, e.g. human or nonhuman primate, a dog, a cat, a horse, a cow, or a rodent. It is also useful for treating damage associated with branch and central vein/artery occlusion, angle-closure glaucoma, open-angle glaucoma (claimed), trauma, edema, age related macular degeneration, retinitis pigmentosa, retinal detachments, damage associated with laser therapy (including photodynamic therapy), and surgical light-induced iatrogenic retinopathy.

ADVANTAGE - The invention produces a response or result favorable to the health or function of a neuron, of a part of the nervous system, or of the nervous system generally.

☐ 3. Document ID: US 20020160933 A1

L5: Entry 3 of 9 File: DWPI Oct 31, 2002

DERWENT-ACC-NO: 2003-328371
DERWENT-WEEK: 200331

TITLE: Producing neurosalutary effect, and treating neurological disorder, in a subject, by administering a therapeutically effective amount of a compound that modulates the activity of N-kinase, to the subject

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2001US-0949200 (September 7, 2001), 2000US-0656915 (September 7, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 20020160933 A1	October 31, 2002		020	A61K031/00

INT-CL (IPC): A61 K 31/00

ABSTRACTED-PUB-NO: US20020160933A
BASIC-ABSTRACT:

NOVELTY - Producing (M1) a neurosalutary effect in a subject, and treating a subject suffering from neurological disorder, involves administering a therapeutically effective amount of a compound (I) that modulates the activity of N-kinase, to the

subject.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) identifying (M2) a compound capable of producing a neurosalutary effect in a subject, by contacting N-kinase or its biologically active fragment, with a test compound and determining the ability of the test compound to modulate the activity of N-kinase;

(2) a compound capable of producing a neurosalutary effect in a subject identified by the above method;

(3) an isolated N-kinase polypeptide (II) of the type that:

(a) is present in neonatal brain tissue

(b) is inhibited in the presence of 6-thioguanine

(c) is activated in the presence of Mn+2 but not by Mg+2 or Ca+2

(d) has a molecular weight of 49 kDa, and

(e) is eluted from a Cibacron Blue column at a NaCl concentration of 1.5-1.75 M;

(4) an antibody which is specifically reactive with an epitope of (II);

(5) a fragment of (II) comprising at least 15 contiguous amino acids, and capable of eliciting an immune response; and

(6) an isolated nucleic acid molecule (III) encoding a polypeptide comprising a sequence of 272 amino acids fully defined in the specification.

ACTIVITY - Anticonvulsant; Cerebroprotective; Neuroprotective; Nootropic.

No supporting biological data is given.

MECHANISM OF ACTION - Modulator of N-kinase activity (claimed); Promotes neuronal survival, axonal outgrowth and neuronal regeneration; Intracellular mediator of axonal outgrowth.

No supporting biological data is given.

USE - M1 is useful for producing a neurosalutary effect, and thus for treating a subject e.g. mammal, preferably human, suffering from neurological disorder such as spinal cord injury (including monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia), epilepsy, stroke and Alzheimer's disease. The treatment method further involves making a first assessment of a nervous system function prior to administering (I) and making a second assessment of a nervous system function after administering (I) to the subject. The nervous system function is a sensory function, cholinergic innervation or vestibulomotor function (claimed).

(II) is useful as bait protein in a two- or three-hybrid assay, to identify other proteins, which bind to or interact with N-kinase.

☐ 4. Document ID: JP 2004523470 W, WO 200220056 A2, AU 200187118 A, EP 1315514 A2

L5: Entry 4 of 9 File: DWPI Aug 5, 2004

DERWENT-ACC-NO: 2002-393816
DERWENT-WEEK: 200451

TITLE: Producing a neurosalutary effect in a subject e.g., one suffering from neurological disorder such as stroke, to treat the subject, by administering a compound that modulates activity of N-kinase

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2000US-0656915 (September 7, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2004523470 W	August 5, 2004		077	A61K045/00
WO 200220056 A2	March 14, 2002	E	042	A61K045/00
AU 200187118 A	March 22, 2002		000	A61K045/00
EP 1315514 A2	June 4, 2003	E	000	A61K038/18

INT-CL (IPC): A61 K 9/10; A61 K 9/127; A61 K 38/18; A61 K 45/00; A61 P 9/10; A61 P 9/12; A61 P 25/00; A61 P 25/02; A61 P 25/08; A61 P 25/14; A61 P 25/16; A61 P 25/18; A61 P 25/24; A61 P 25/28; A61 P 43/00; C07 K 14/475; C07 K 16/40; C12 N 9/12; C12 N 15/09; C12 Q 1/48; G01 N 33/15; G01 N 33/50; G01 N 33/53; G01 N 33/566

ABSTRACTED-PUB-NO: WO 200220056A
BASIC-ABSTRACT:

NOVELTY - Producing (M1) a neurosalutary effect in a subject e.g., a subject suffering from a neurological disorder, to treat the subject suffering from the neurological disorder, involving administering to the subject a compound (I) that modulates the activity of N-kinase, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated N-kinase polypeptide (II) of the type that: is present in neonatal brain tissue; is inhibited in the presence of 6-thioguanine; is activated in the presence of Mn2+, but not by Mg2+ or Ca2+; has a molecular weight of approximately 49 kDa; and is eluted from a Cibacron Blue column at a sodium chloride concentration of 1.5-1.75 M;

(2) an antibody (III) which is specifically reactive with an epitope of (II);

(3) a fragment (IV) of (I), which comprises at least 15 contiguous amino acids, and is able to elicit an immune response;

(4) an isolated nucleic acid molecule that encodes (II); and

(5) a compound capable of producing a neurosalutary effect in a subject identified using (II).

ACTIVITY - Nootropic; neuroprotective; cerebroprotective; anticonvulsant; vulnerary; tranquilizer; antiparkinsonian; antimanic; antidepressant.

MECHANISM OF ACTION - N-kinase activity modulator; neuronal survival modulator; neuronal regeneration modulator; neuronal axonal outgrowth of central nervous system neurons e.g., retinal ganglion cells, modulator (all claimed).

No data given.

USE - (I) is useful for producing a neurosalutary effect in a subject e.g., a subject suffering from a neurological disorder, to treat the subject (preferably, humans)

suffering from the neurological disorder. The neurosalutary effect is produced by modulating neuronal survival, modulating neuronal regeneration or modulating neuronal axonal outgrowth of central nervous system neurons e.g., retinal ganglion cells, in a subject suffering from a neurological disorder such as spinal cord injury characterized by monoplegia, diplegia, paraplegia, hemoplegia and quadriplegia, or suffering from epilepsy, stroke or Alzheimer's disease.

(II) is useful for identifying a compound capable of producing a neurosalutary effect in a subject, preferably a compound which inhibits or stimulates the activity of N-kinase, which involves contacting (II) or its biologically active fragment with a test compound and determining the ability of the test compound to modulate the activity of N-kinase, thereby identifying a compound capable of producing a neurosalutary effect in a subject. The ability of the test compound to modulate the activity of N-kinase is determined by assessing the ability of the test compound to modulate N-kinase-dependant phosphorylation of a substrate. Optionally, (I) is identified using (II) by the following method which involves contacting (II) or its biologically active fragment, with a test compound, an N-kinase substrate (e.g., histone HF-1 protein), radioactive ATP (preferably gamma -32P), and Mn2+; and determining the ability of the test compound to modulate N-kinase dependent phosphorylation of the substrate, thereby identifying a compound capable of producing a neurosalutary effect in a subject. (II) used in the methods described above is preferably a recombinantly produced human N-kinase. Optionally, (II) is bovine N-kinase purified from a bovine source. The methods further involve determining the ability of the test compound to modulate axonal outgrowth of central nervous system neuron (all claimed).

(M1) is useful for treating a neurological disorder such as dementia's related to Alzheimer's disease, Parkinson's disease, senile dementia, Huntington's disease, Creutzfeldt-Jakob disease, Korsakoff's psychosis, mania, anxiety disorders, obsessive-compulsive disorder, anxiety, bipolar affective disorder. The methods are useful for preventing or treating neurological deficits in embryos or fetuses in utero, in premature infants, or in children with need of such treatment, including those with neurological birth defects. (I) is also useful for modulating activity of N-kinase, in vitro to modulate axonal outgrowth in vitro.

☐ 5. Document ID: WO 200206341 A1, AU 200180566 A

L5: Entry 5 of 9 File: DWPI Jan 24, 2002

DERWENT-ACC-NO: 2002-291790
DERWENT-WEEK: 200236

TITLE: Isolated neurotrophic factor useful for treating neurological conditions is present in a medium containing Schwann cells culture

INVENTOR: BENOWITZ, L I ; IRWIN, C A ; JACKSON, P

PRIORITY-DATA: 2000US-0616287 (July 14, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200206341 A1	January 24, 2002	E	050	C07K014/47
AU 200180566 A	January 30, 2002		000	C07K014/47

INT-CL (IPC): C07 K 14/47

ABSTRACTED-PUB-NO: WO 200206341A
BASIC-ABSTRACT:

NOVELTY - An isolated neurotrophic factor (I) of the type that is present in a medium containing Schwann cells culture is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) a method for producing a neurosalutary effect in a subject by administering the neurotrophic factor (I) to the subject; and

(2) a method for treating neurological disorder involving administering (I) to a subject suffering from the neurological disorder.

ACTIVITY - Anticonvulsant; Nootropic; Neuroprotective; Cerebroprotective; Tranquilizer; Vulnerary; Neuroleptic; Antidepressant; Antidiabetic; Antiparkinsonian; Antimanic; Hypotensive; Analgesic; Antibacterial; Antiinflammatory; Antipyretic; Anti-HIV.

MECHANISM OF ACTION - Axonal outgrowth of naive goldfish retinal ganglion cells stimulator; Axonal outgrowth of embryonic rat spinal cord neuron stimulator; Modulators of neuronal survival, neuronal regeneration and neuronal axonal outgrowth of central nervous system neurons such as retinal ganglion cells.

USE - For producing neurosalutory effect in a subject such as mammal e.g. human suffering from a neurological disorder such as spinal cord injury, e.g. monoplegia, diplegia, paraplegia, hemiplegia, or quadriplegia, epilepsy e.g. posttraumatic epilepsy, Alzheimer's disease (all claimed). The neurological disorders include traumatic or toxic injuries to peripheral or cranial nerves, traumatic brain injury, stroke, cerebral aneurism, cognitive and neurodegenerative disorders such as dementias, Huntington's disease, Gilles de la Tourette's syndrome, multiple sclerosis, amyotrophic, lateral sclerosis, hereditary motor and sensory neuropathy (Charcot-Marie-Tooth disease), diabetic neuropathy, progressive supranuclear palsy, Jakob-Creutzfeldt disease or disorders included in Harrison's Principles of Internal Medicine (Braunwald et-al.McGraw-Hill,2001) and in the American Psychiatric Association's Diagmestic and statistical manual of mental Disorders DSM-IV (American Psychiatric Press,2000); for treating hypertension and sleep disorders, neuropsychiatric disorders such as depression, schizophrenia, schizoaffective disorder, korsakoff's psychosis, mania, anxiety disorders, or phobic disorder, learning or memory disorders (such as amnesia and age-related memory loss), attention deficit disorder, dysthymic disorder, major depressive disorder, mania, obsessive compulsive disorder, psychoactive substance, use disorder, panic disorder, bipolar affective disorder, psychogenic pain syndromes, and eating disorders; for treating injuries of nervous system due to an infections disease (such as meningitis, high fever of various etiologies, HIV, syphilis, or post-polio syndrome) or due to electricity (including contact with electricity or lightening and complications from electro-convulsive psychiatric therapy); for preventing or treating neurological deficits in embryos or fetuses in utero, in premature infants, or in children with need of such treatment, including those with neurological birth defects.

ADVANTAGE - The formulation provides sustained delivery of (I) for at least one-week (preferably at least one month) after the formulation is administered to the subject. The neurotrophic factor stimulates axonal outgrowth of naive goldfish retinal ganglion cells, embryonic rat spinal cord neurons and passes through a centrifugal filter with a 1 kDa cut-off. The neurotrophic factor further fails to bind to a 18C reversed-phase HPLC column, forms a compound that elutes from a reverse-phase HPLC column, at 23 minutes, after being chemically derivatized with AQC and has an elution time of 6 minutes on a G10-Sepharose size-exclusive column.

☐ 6. Document ID: JP 2003534385 W, WO 200191783 A2, AU 200168147 A, US 20020119923 A1, EP 1289540 A2

L5: Entry 6 of 9 File: DWPI Nov 18, 2003

DERWENT-ACC-NO: 2002-097736
DERWENT-WEEK: 200401

TITLE: Method of producing a neurosalutary effect in a subject with a neurological condition, comprises administering a macrophage derived factor

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 2000US-208778P (June 1, 2000), 2001US-0872347 (June 1, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2003534385 W	November 18, 2003		045	A61K035/26
WO 200191783 A2	December 6, 2001	E	035	A61K038/18
AU 200168147 A	December 11, 2001		000	A61K038/18
US 20020119923 A1	August 29, 2002		000	A61K038/18
EP 1289540 A2	March 12, 2003	E	000	A61K038/18

INT-CL (IPC): A61 K 9/10; A61 K 9/12; A61 K 9/127; A61 K 31/00; A61 K 31/45; A61 K 31/7076; A61 K 31/7105; A61 K 31:00; A61 K 35/26; A61 K 38/18; A61 K 38/22; A61 K 38:18; A61 K 45/00; A61 P 25/00; A61 P 25/08; A61 P 25/28; A61 P 43/00; A61 K 38/18; A61 K 31:00

ABSTRACTED-PUB-NO: US20020119923A
BASIC-ABSTRACT:

NOVELTY - A method of producing a neurosalutary effect in a subject with a neurological condition comprises administering a macrophage-derived factor.

DETAILED DESCRIPTION - A method of producing a neurosalutary effect in a subject with a neurological condition comprises administering a macrophage-derived factor, and optionally a cAMP modulator or an axogenic factor. INDEPENDENT CLAIMS are included for:

(1) compositions for producing a neurosalutary effect comprising a macrophage-derived factor and optionally a cAMP modulator or an axogenic factor;

(2) a method comprising the administration of oncomodulin, and for producing a neurosalutary effect with an effective amount of AF-1; and

(3) a composition comprising macrophage-derived factor and carrier packed with instructions for use of a pharmaceutical composition.

ACTIVITY - Anticonvulsant; nootropic; neuroprotective; antiparkinsonian; nootropic; anticonvulsant; neuroleptic; antidiabetic; antidepressant; tranquilizer.

No specific biological data given.

MECHANISM OF ACTION - Neuronal survival modulator; neuronal regeneration modulator; neuronal axonal outgrowth modulator.

USE - For treating neurological disorders, e.g. spinal cord injury, such as monoplegia, diplegia, paraplegia, hemiplegia or quadriplegia; epilepsy, such as posttraumatic epilepsy; or Alzheimer's disease (claimed), also Parkinson's disease,

senile dementia, Huntington's disease, Gilles de la Tourette's syndrome, multiple sclerosis, amyotrophic lateral sclerosis, hereditary motor and sensory neuropathy, diabetic neuropathy, progressive supranuclear palsy, Creutzfeldt-Jakob disease, depression, schizophrenia, schizoaffective disorder, Korsakoff's psychosis, mania, anxiety disorders, phobic disorders, learning or memory disorders, attention deficit disorder, dysthymic disorder, major depressive disorder, mania, obsessive-compulsive disorder, psychoactive substance use disorder, anxiety, phobias, panic disorder, bipolar affective disorder, psychogenic pain syndromes and eating disorders.
ABSTRACTED-PUB-NO:

WO 200191783A EQUIVALENT-ABSTRACTS:

NOVELTY - A method of producing a neurosalutary effect in a subject with a neurological condition comprises administering a macrophage-derived factor.

DETAILED DESCRIPTION - A method of producing a neurosalutary effect in a subject with a neurological condition comprises administering a macrophage-derived factor, and optionally a cAMP modulator or an axogenic factor. INDEPENDENT CLAIMS are included for:

(1) compositions for producing a neurosalutary effect comprising a macrophage-derived factor and optionally a cAMP modulator or an axogenic factor;

(2) a method comprising the administration of oncomodulin, and for producing a neurosalutary effect with an effective amount of AF-1; and

(3) a composition comprising macrophage-derived factor and carrier packed with instructions for use of a pharmaceutical composition.

ACTIVITY - Anticonvulsant; nootropic; neuroprotective; antiparkinsonian; nootropic; anticonvulsant; neuroleptic; antidiabetic; antidepressant; tranquilizer.

No specific biological data given.

MECHANISM OF ACTION - Neuronal survival modulator; neuronal regeneration modulator; neuronal axonal outgrowth modulator.

USE - For treating neurological disorders, e.g. spinal cord injury, such as monoplegia, diplegia, paraplegia, hemiplegia or quadriplegia; epilepsy, such as posttraumatic epilepsy; or Alzheimer's disease (claimed), also Parkinson's disease, senile dementia, Huntington's disease, Gilles de la Tourette's syndrome, multiple sclerosis, amyotrophic lateral sclerosis, hereditary motor and sensory neuropathy, diabetic neuropathy, progressive supranuclear palsy, Creutzfeldt-Jakob disease, depression, schizophrenia, schizoaffective disorder, Korsakoff's psychosis, mania, anxiety disorders, phobic disorders, learning or memory disorders, attention deficit disorder, dysthymic disorder, major depressive disorder, mania, obsessive-compulsive disorder, psychoactive substance use disorder, anxiety, phobias, panic disorder, bipolar affective disorder, psychogenic pain syndromes and eating disorders.

☐ 7. Document ID: EP 1466606 A2, WO 9911274 A1, AU 9866568 A, EP 1009412 A1, CN 1286632 A, KR 2001023578 A, JP 2001516695 W, US 20020042390 A1, US 20020055484 A1, AU 748961 B, US 6440455 B1, US 20020128223 A1, US 20020137721 A1, NZ 503073 A, US 6551612 B2, RU 2212241 C2, US 20040014710 A1, EP 1009412 B1, DE 69825292 E

L5: Entry 7 of 9 File: DWPI Oct 13, 2004

DERWENT-ACC-NO: 1999-228934
DERWENT-WEEK: 200467

TITLE: Modulating axonal outgrowth of central nervous system

INVENTOR: BENOWITZ, L I

PRIORITY-DATA: 1997US-0921902 (September 2, 1997), 2001US-0997688 (November 29, 2001), 2001US-0997687 (November 29, 2001), 2002US-0145224 (May 14, 2002), 2002US-0144952 (May 14, 2002), 2003US-0385031 (March 10, 2003)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 1466606 A2	October 13, 2004	E	000	A61K031/52
WO 9911274 A1	March 11, 1999	E	043	A61K031/70
AU 9866568 A	March 22, 1999		000	
EP 1009412 A1	June 21, 2000	E	000	
CN 1286632 A	March 7, 2001		000	A61K031/70
KR 2001023578 A	March 26, 2001		000	A61K031/70
JP 2001516695 W	October 2, 2001		047	A61K031/708
US 20020042390 A1	April 11, 2002		000	A61K031/708
US 20020055484 A1	May 9, 2002		000	A61K031/7105
AU 748961 B	June 13, 2002		000	A61K031/70
US 6440455 B1	August 27, 2002		000	A61K009/127
US 20020128223 A1	September 12, 2002		000	A61K031/708
US 20020137721 A1	September 26, 2002		000	A61K031/708
NZ 503073 A	November 22, 2002		000	A61K031/70
US 6551612 B2	April 22, 2003		000	A61K009/127
RU 2212241 C2	September 20, 2003		000	A61K031/70
US 20040014710 A1	January 22, 2004		000	A61K031/7076
EP 1009412 B1	July 28, 2004	E	000	A61K031/70
DE 69825292 E	September 2, 2004		000	A61K031/70

6551612 B2 , RU 2212241 C2 , US 20040014710 A1 INT-CL (IPC): A61 K 9/127; A61 K 9/16; A61 K 31/52; A61 K 31/522; A61 K 31/70; A61 K 31/7076; A61 K 31/708; A61 K 31/7105; A61 P 25/00; A61 P 25/08; A61 P 43/00; C07 H 19/167

ABSTRACTED-PUB-NO: US 6440455B
BASIC-ABSTRACT:

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or

inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.
ABSTRACTED-PUB-NO:

US20020042390A EQUIVALENT-ABSTRACTS:

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.

US20020055484A

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls

then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.

US20020128223A

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.

US20020137721A

NOVELTY - Stimulating axonal outgrowth following injury, inhibiting axonal outgrowth in patients suffering or prone to suffering from a condition characterized by increased axonal outgrowth, or modulating axonal outgrowth of central nervous system neurons (CNSN) comprises contacting the CNSN with a composition comprising a purine nucleoside (I) or its analogue.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) use of (I) in the manufacture of a medicament for modulating axonal outgrowth of mammalian central nervous system neurons; and

(2) a packed formulation comprising a composition comprising inosine (Ia), guanosine (Ib) or 6-thioguanine (Ic) and a carrier with instructions for use for treatment of CNS disorders.

ACTIVITY - Neuronal; Central nervous system

MECHANISM OF ACTION - Modulating axonal outgrowth of central nervous system neurons.

USE - To modulate axonal outgrowth of CNS neurons, preferably stimulating or

inhibiting the outgrowth. The method is particularly useful for stimulating axonal outgrowth following injury such as that due to stroke, traumatic brain injury, cerebral aneurysm, spinal cord injury (preferably monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia) or for inhibiting axonal outgrowth in CNS neurons in patients suffering or prone to suffering from conditions characterized by increased axonal outgrowth of CNS neurons such as epilepsy, especially posttraumatic epilepsy, neuropathic pain syndrome. The method is useful in mammals, preferably humans, particularly for modulation of growth of CNS retinal ganglion cells.

DESCRIPTION OF DRAWING(S) - The figure shows the quantitation of purinergic effects on axonal growth. It shows the axonal growth in response to adenosine (A), guanosine (G), cytidine (C), uridine (U) and thymidine (T) at a concentration of 1, 10 and 100 mu M. Data are normalized by subtracting the level of growth in the negative controls then dividing by the net growth in positive controls treated with 20-30% AF-1. EC50 values estimated from these data are 10-15 micro M for A and 20-30 micro M for G.

WO 9911274A

☐ 8. Document ID: WO 9606859 A1, CN 1164858 A, AU 9535393 A, EP 777686 A1, NZ 293048 A, JP 10505238 W, KR 97705577 A, US 5898066 A, AU 713028 B, AU 200012463 A, RU 2157223 C2

L5: Entry 8 of 9 File: DWPI Mar 7, 1996

DERWENT-ACC-NO: 1996-160307
DERWENT-WEEK: 200148

TITLE: Isolated neurotrophic polypeptide(s) - useful for inducing axonal extension in neuronal cells, partic. for treating nervous tissue damage

INVENTOR: BENOWITZ, L I ; IRWIN, C A ; JACKSON, P

PRIORITY-DATA: 1994US-0296661 (August 26, 1994), 2000AU-0012463 (January 18, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9606859 A1	March 7, 1996	E	076	C07K014/475
CN 1164858 A	November 12, 1997		000	C07K014/475
AU 9535393 A	March 22, 1996		000	C07K014/475
EP 777686 A1	June 11, 1997	E	000	C07K014/475
NZ 293048 A	January 26, 1998		000	C07K014/475
JP 10505238 W	May 26, 1998		062	C12N015/09
KR 97705577 A	October 9, 1997		000	C07K014/475
US 5898066 A	April 27, 1999		000	C07K002/00
AU 713028 B	November 18, 1999		000	C07K014/475
AU 200012463 A	July 6, 2000		000	C07K007/06
RU 2157223 C2	October 10, 2000		000	A61K035/60

INT-CL (IPC): A61 K 35/60; A61 K 38/00; A61 K 38/22; C07 K 2/00; C07 K 7/06; C07 K 7/08; C07 K 14/435; C07 K 14/475; C07 K 14/48; C07 K 14/52; C07 K 16/22; C12 N 15/09; C12 N 15/12; C12 P 21/02; C12 P 21/08; C12 Q 1/68

ABSTRACTED-PUB-NO: US 5898066A

BASIC-ABSTRACT:

(A) A neurotrophic polypeptide (NP) is claimed which is selected from: (a) a polypeptide having a mol.wt. of about 700 D by mass spectroscopy, which can be isolated from medium in which glial sheath cells have been cultured by mol. wt. sepn., two-phase extn. and reversed phase HPLC, which retains activity after heating at 95 deg.C for < 15 mins., which retains activity after digestion with pronase and trypsin, and is hydrophilic; and (b) a protein having a mol.wt. of about 12 000 D which can be isolated from medium in which glial sheath cells have been cultured by mol.wt. sepn. and anion exchange chromatography, where the protein binds to the column at pH 10 but not at pH 8.4 and can be eluted with 0.2 M NaCl, loses activity upon heating at 95 deg.C for 15 mins. and loses activity following digestion with trypsin or proteinase K. Also claimed are: (B) a nucleotide sequence encoding a NP as in (A); and (C) an antibody immunoreactive with a NP as in (A).

USE - The NPs can be used for inducing axonal extension in neuronal cells (claimed). They can be used in the treatment of optic nerve, brain, spinal cord and other nervous tissue damage. They can be used for treating e.g. trauma damage, demyelinating diseases, autoimmune disorders or degenerative diseases. The prods. can also be used for the screening of drugs which modulate the activity and/or the expression of the NPs and in screening of patient samples for the presence of functional NPs.
ABSTRACTED-PUB-NO:

WO 9606859A EQUIVALENT-ABSTRACTS:

(A) A neurotrophic polypeptide (NP) is claimed which is selected from: (a) a polypeptide having a mol.wt. of about 700 D by mass spectroscopy, which can be isolated from medium in which glial sheath cells have been cultured by mol. wt. sepn., two-phase extn. and reversed phase HPLC, which retains activity after heating at 95 deg. C for < 15 mins., which retains activity after digestion with pronase and trypsin, and is hydrophilic; and (b) a protein having a mol.wt. of about 12 000 D which can be isolated from medium in which glial sheath cells have been cultured by mol.wt. sepn. and anion exchange chromatography, where the protein binds to the column at pH 10 but not at pH 8.4 and can be eluted with 0.2 M NaCl, loses activity upon heating at 95 deg. C for 15 mins. and loses activity following digestion with trypsin or proteinase K. Also claimed are: (B) a nucleotide sequence encoding a NP as in (A); and (C) an antibody immunoreactive with a NP as in (A).

USE - The NPs can be used for inducing axonal extension in neuronal cells (claimed). They can be used in the treatment of optic nerve, brain, spinal cord and other nervous tissue damage. They can be used for treating e.g. trauma damage, demyelinating diseases, autoimmune disorders or degenerative diseases. The prods. can also be used for the screening of drugs which modulate the activity and/or the expression of the NPs and in screening of patient samples for the presence of functional NPs.

Items 1 - 11 of 11 One p

1: Cook DJ, Kulbatski I, Tator CH. Related Articles, Li

Inosine reverses the inhibitory effects of the L-type Ca2+ channel antagonist, DM-BODIPY-dihydropyridine, on neuritogenesis in an in vitro rat superior cervical ganglia axotomy model.
Neurosci Lett. 2004 Mar 25;358(2):75-8.
PMID: 15026152 [PubMed - indexed for MEDLINE]

2: Benowitz LI, Goldberg DE, Irwin N. Related Articles, Li

Inosine stimulates axon growth in vitro and in the adult CNS.
Prog Brain Res. 2002;137:389-99. Review.
PMID: 12440381 [PubMed - indexed for MEDLINE]

3: Sillibourne JE, Milne DM, Takahashi M, Ono Y, Meek DW. Related Articles, Li

Centrosomal anchoring of the protein kinase CK1delta mediated by attachmer to the large, coiled-coil scaffolding protein CG-NAP/AKAP450.
J Mol Biol. 2002 Sep 27;322(4):785-97.
PMID: 12270714 [PubMed - indexed for MEDLINE]

4: Benowitz LI, Goldberg DE, Irwin N. Related Articles, Li

A purine-sensitive mechanism regulates the molecular program for axon grow
Restor Neurol Neurosci. 2001;19(1-2):41-9. Review.
PMID: 12082228 [PubMed - indexed for MEDLINE]

5: Spiegelman VS, Stavropoulos P, Latres E, Pagano M, Ronai Z, Slaga TJ, Fuchs SY. Related Articles, Li

Induction of beta-transducin repeat-containing protein by JNK signaling and i role in the activation of NF-kappaB.
J Biol Chem. 2001 Jul 20;276(29):27152-8. Epub 2001 May 24.
PMID: 11375388 [PubMed - indexed for MEDLINE]

6: Haydar TF, Kuan CY, Flavell RA, Rakic P. Related Articles, Li

The role of cell death in regulating the size and shape of the mammalian forebrain.
Cereb Cortex. 1999 Sep;9(6):621-6. Review.
PMID: 10498280 [PubMed - indexed for MEDLINE]

7: Fuchs SY, Dolan L, Davis RJ, Ronai Z. Related Articles, Li

Phosphorylation-dependent targeting of c-Jun ubiquitination by Jun N-kinase.
Oncogene. 1996 Oct 3;13(7):1531-5.
PMID: 8875991 [PubMed - indexed for MEDLINE]

8: Adler V, Pincus MR, Posner S, Upadhyaya P, El-Bayoumy K, Ronai Z. Related Articles, Li

Effects of chemopreventive selenium compounds on Jun N-kinase activities.
Carcinogenesis. 1996 Sep;17(9):1849-54.
PMID: 8824505 [PubMed - indexed for MEDLINE]

9: Boniece IR, Wagner JA. Related Articles, Li

h cb h g e e e fcg

NGF protects PC12 cells against ischemia by a mechanism that requires the N kinase.
J Neurosci Res. 1995 Jan 1;40(1):1-9.
PMID: 7714916 [PubMed - indexed for MEDLINE]

10: Estruch F, Treitel MA, Yang X, Carlson M. Related Articles, Li

N-terminal mutations modulate yeast SNF1 protein kinase function.
Genetics. 1992 Nov;132(3):639-50.
PMID: 1468623 [PubMed - indexed for MEDLINE]

11: Rowland-Gagne E, Greene LA. Related Articles, Li

Multiple pathways of N-kinase activation in PC12 cells.
J Neurochem. 1990 Feb;54(2):423-33.
PMID: 2153751 [PubMed - indexed for MEDLINE]

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1: J Neurochem. 1990 Feb;54(2):423-33.

Multiple pathways of N-kinase activation in PC12 cells.

Rowland-Gagne E, Greene LA.

Department of Pharmacology, New York University School of Medicine.

Past work established a cell-free assay for a nerve growth factor (NGF)-activat protein kinase activity (designated N-kinase) that utilizes tyrosine hydroxylase and histone H1 as substrates and that is distinct from a variety of well-characterized kinases. This study explores the specificity and mechanistic pathway(s) by which N-kinase activity is regulated in PC12 rat pheochromocytoma cells. N-kinase is rapidly activated in these cells by treatm with NGF, epidermal growth factor (EGF), basic fibroblast growth factor (bFC phorbol ester, or dibutyryl cyclic AMP. Our data indicate that the stimulated activity is the same for each agent by several criteria: It exhibits the same characteristic biphasic elution pattern by Mono S fast protein liquid chromatography (FPLC), except for the case of dibutyryl cyclic AMP in which one of the activity peaks is somewhat shifted; it shows the same elution pattern FPLC on a Superose 12 column; it possesses identical substrate specificity; an except in the case of dibutyryl cyclic AMP, it does not show additivity when e agent is added simultaneously with NGF. The multiple forms of N-kinase are interconvertible in that rechromatography on a Mono S column yields a single peak of activity. Also, when NGF and dibutyryl cyclic AMP are simultaneousl presented to cells, the chromatographic profile resembles that with NGF alone Activation occurs through several independent initial pathways. Down-regulat of protein kinase C by phorbol ester pretreatment prevents N-kinase activation phorbol ester, but not by the other agents. A PC12 cell-derived line deficient in cyclic AMP-dependent protein kinase II activity exhibits N-kinase activation b all treatments except dibutyryl cyclic AMP. The properties of N-kinase sugges that it is similar or identical to the ribosomal S6 protein kinase described by Blenis and Erikson. Additional experiments revealed that N-kinase activity can stimulated in several cell lines in addition to PC12 cells. These findings indica that the N-kinase can be activated via multiple second-messenger pathways an that it could therefore potentially play a significant role in mediating shared intracellular responses to various extracellular signals.

PMID: 2153751 [PubMed - indexed for MEDLINE]

h cb hg e e e fcg c e e e b b e b c

1: Genetics. 1992 Nov;132(3):639-50.

N-terminal mutations modulate yeast SNF1 protein kinase functi

Estruch F, Treitel MA, Yang X, Carlson M.

Department of Genetics and Development, Columbia University, College of Physicians and Surgeons, New York, New York 10032.

The SNF1 protein kinase is required for expression of glucose-repressed genes response to glucose deprivation. The SNF4 protein is physically associated wit SNF1 and positively affects the kinase activity. We report here the characterization of a dominant mutation, SNF1-G53R, that was isolated as a suppressor of the requirement for SNF4. The mutant SNF1-G53R protein is sti responsive to SNF4 but has greatly elevated kinase activity in immune comple assays; in contrast, the activity is wild type in a protein blot assay. Deletion of region N-terminal to the kinase domain (codons 5-52) reduces kinase activity i vitro, but the mutant SNF1-delta N kinase is still dependent on SNF4. The N terminus is not required for the regulatory response to glucose. In gel filtration chromatography, the SNF1, SNF1-G53R and SNF1-delta N protein showed different elution profiles, consistent with differential formation of high molecu weight complexes. Taken together, the results suggest that the N terminus positively affects the function of the SNF1 kinase and may be involved in interaction with a positive effector other than SNF4. We also showed that the conserved threonine residue 210 in subdomain VIII, which is a phosphorylatio site in other kinases, is essential for SNF1 activity. Finally, we present evidenc that when the C terminus is deleted, overexpression of the SNF1 kinase domai deleterious to the cell.

PMID: 1468623 [PubMed - indexed for MEDLINE]

h cb hg e e e fcg c e e e b b e b c

1: J Neurosci Res. 1995 Jan 1;40(1):1-9.

NGF protects PC12 cells against ischemia by a mechanism that requires the N-kinase.

Boniece IR, Wagner JA.

Department of Neurology and Neuroscience, Cornell University Medical Colle New York, New York 10021.

Nerve growth factor (NGF), which has been shown to act as a morphological a neurochemical differentiating factor in PC12 cells, also protects PC12 cells fro the toxicity of serum withdrawal and ischemia. By using a previously establish in vitro model of ischemia, which incorporates the combination of anoxia with glucose deprivation (Boniece and Wagner: J Neurosci 13:4220-4228, 1993), w have been able to study the signal transduction pathways upon which NGF-induced survival is dependent. Here we demonstrate that inhibitors of the N-kinase and NGF-induced neuritogenesis, 6-thioguanine and 2-aminopurine, prevent the protective effects of NGF, while they have little, if any, effect on th protection conferred by epidermal growth factor (EGF) or dbcAMP. This suggests that only NGF acts by a mechanism that depends strongly on the N-kinase. Furthermore, the methyltransferase inhibitor 5'-deoxy-5'-methylthioadenosine (MTA), which also inhibits NGF-induced neuritogenesis, inhibits the protective effect of NGF but not the protective effects of EGF or dbcAMP. Thus, the neuroprotective effect of NGF requires some of the same signal transduction steps used by NGF to promote differentiation and neurite formation. Furthermore, we found that exposure of PC12 cells to retinoic acid, which promotes the differentiation and inhibits the growth of PC12 cells, also improves cell survival during ischemia. In addition, a combination of NGF and retinoic acid was more effective than either agent alone. It is likely that these two agents confer protection by independent pathways.

PMID: 7714916 [PubMed - indexed for MEDLINE]

h cb h g e e e fcg c e e e b b e b c

1: Carcinogenesis. 1996 Sep;17(9):1849-54. Related Articles, L

Effects of chemopreventive selenium compounds on Jun N-kinase activities.

Adler V, Pincus MR, Posner S, Upadhyaya P, El-Bayoumy K, Ronai Z.

Molecular Carcinogenesis Program, American Health Foundation, Valhalla, N 10595, USA.

Activation of Jun-N-kinases (JNK) is stimulated by diverse agents including U irradiation, heat shock, tumor necrosis factor and osmotic shock. In the present study we have elucidated the effect of the organoselenium chemopreventive ag 1,4-phenylenebis(methylene)selenocyanate (p-XSC, on UV-mediated JNK activation. Using mouse fibroblasts as a model cell system we found that low concentrations (1-10 microM range) of p-XSC did not affect JNK activity, yet were capable of potentiating JNK activity when administered prior to UV-irradiation. While higher doses of p-XSC have minimal effect on JNK activati when combined with UV, there is a dose-dependent decrease in JNK activatio Similar to its effects on JNK, p-XSC is a potent inducer of src-related tyrosine kinases. p-XSC mediated changes in JNK activation correlate with its ability t potentiate the association of JNK with p21ras, in a manner similar to that we h previously observed with GTP or sodium vanadate. That p-XSC can modulate JNK activities points to a possible mechanism by which it contributes to the ce ability to cope with stress.

PMID: 8824505 [PubMed - indexed for MEDLINE]

h cb hg e e e fcg c e e e b b e b c

1: Oncogene. 1996 Oct 3;13(7):1531-5. Related Articles, l

Phosphorylation-dependent targeting of c-Jun ubiquitination by Jun N-kinase.

Fuchs SY, Dolan L, Davis RJ, Ronai Z.

Molecular Carcinogenesis Program, American Health Foundation, Valhalla, N York 10595, USA.

Ubiquitination of key cellular regulatory proteins marks them for efficient degradation via the proteasome pathway. The delta domain of c-jun is essentia for its ubiquitination and also for the activating phosphorylation of neighborin serines by the stress activated jun-N-terminal kinases (JNK). Using an in vitro model system we demonstrate that JNK is among the hydrophobic binding proteins that target c-jun for efficient ubiquitination. Immunodepletion of JNK markedly inhibits c-jun ubiquitination. Conversely, c-jun ubiquitination is increased by adding purified JNK2 or extracts prepared from cells transfected with JNK2. Although c-jun ubiquitination is enhanced by JNK, the phosphorylation of c-jun on Ser73 by JNK protects c-jun from ubiquitination a prolongs its half-life. The dual activity of JNK in targeting c-jun for ubiquitination or in protecting c-jun from entering this pathway via phosphorylation points to the role of JNK in the control of c-jun stability in cel exposed to environmental stress or inflammatory cytokines.

PMID: 8875991 [PubMed - indexed for MEDLINE]

h cb hg e e e fcg c e e e b b e b c

1: Cereb Cortex. 1999 Sep;9(6):621-6.

The role of cell death in regulating the size and shape of the mammalian forebrain.

Haydar TF, Kuan CY, Flavell RA, Rakic P.

Section of Neurobiology, Yale University School of Medicine, New Haven, C' 06510, USA. thaydar@kafka.med.yale.edu

The size of the cerebral cortex is determined by the rate of production of neurc and glial cells in the proliferative ventricular and subventricular zones. Recent studies from targeted mutations of different death-effector gene families indica that programmed cell death (PCD) plays an important role in cell production a early morphogenesis of the mammalian forebrain before the formation of neuronal connections. For example, disruption of the c/Jun N-kinase signaling pathway by double-targeted mutation of both Jnk1 and Jnk2 results in increase PCD in the forebrain leading to precocious degeneration of cerebral precursors contrast, disturbance of the caspase cascade by targeted disruption of either ca 9 or casp-3 leads to decreased PCD causing expansion and exencephaly of the forebrain as well as supernumerary neurons in the cerebral cortex. The supernumerary neurons in these knockout mice align radially and form an expanded cortical plate which begins to form cerebral convolutions. Thus, the precise coordination of different apoptotic signaling pathways during early sta of neurogenesis is crucial for regulation of the proper cortical size and shape.

Publication Types:
- Review
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PMID: 10498280 [PubMed - indexed for MEDLINE]

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TI   N-terminal mutations modulate yeast SNF1 protein kinase function.
AU   Estruch, F.; Treitel, M.A.; Yang, X.L.; Carlson, M.
CS   Columbia University, New York, NY
AV   DNAL (442.8 G28)
SO   Genetics,   ***Nov 1992.***   Vol. 132, No. 3. p. 639-650
     Publisher: Baltimore, Md. : Genetics Society of America.
     CODEN: GENTAE; ISSN: 0016-6731
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AN   2002:251992  BIOSIS
DN   PREV200200251992
TI   Apoptosis and JNK activation are differentially regulated by Fas
     expression level in renal tubular epithelial cells (RTC).
AU   Khan, S. [Reprint author]; Koepke, A. [Reprint author]; Jarad, G. [Reprint
     author]; Schlessman, K. [Reprint author]; Wang, B. [Reprint author];
     Konieczkowski, M. [Reprint author]; Schelling, J. [Reprint author]
CS   Case Western Reserve U., Cleveland, OH, USA
SO   Journal of the American Society of Nephrology, (September, 2000) Vol. 11,
     No. Program and Abstract Issue, pp. 458A. print.
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Meeting Info.: 33rd Annual Meeting of the American Society of Nephrology and the 2000 Renal Week. Toronto, Ontario, Canada. October 10-16, 2000. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
T Conference; (Meeting)
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A English
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N 2002:251976 BIOSIS
N PREV200200251976
I Genetic mechanisms and MAPK-signaling in post-infarction heart failure in rats.
U Gurevich, Andrey K. [Reprint author]; Weinberger, Howard D. [Reprint author]; Nemenoff, Raphael A. [Reprint author]; Bedigian, Martin P.; Schrier, Robert W. [Reprint author]
S Department of Medicine, University of Colorado Health Sciences Center, Denver, CO, USA
O Journal of the American Society of Nephrology, (September, 2000) Vol. 11, No. Program and Abstract Issue, pp. 455A. print.
Meeting Info.: 33rd Annual Meeting of the American Society of Nephrology and the 2000 Renal Week. Toronto, Ontario, Canada. October 10-16, 2000. American Society of Nephrology.
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N 1999:483463 BIOSIS
N PREV199900483463
I The role of cell death in regulating the size and shape of the mammalian forebrain.
U Haydar, Tarik F. [Reprint author]; Kuan, Chia-Yi [Reprint author]; Flavell, Richard A.; Rakic, Pasko [Reprint author]
S Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA
O Cerebral Cortex, (Sept., 1999) Vol. 9, No. 6, pp. 621-626. print.
ISSN: 1047-3211.
T Article
A English
D Entered STN: 16 Nov 1999
Last Updated on STN: 16 Nov 1999

3 ANSWER 5 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
N 1999:46474 BIOSIS
N PREV199900046474
I Expression of GAP-43 is regulated by multiple pathways in PC12 cells.
U Burry, R. W. [Reprint author]
S Div. Neurosi. Graduate Program, Ohio State Univ., Columbus, OH 43210, USA
O Society for Neuroscience Abstracts, (1998) Vol. 24, No. 1-2, pp. 543. print.
Meeting Info.: 28th Annual Meeting of the Society for Neuroscience, Part 1. Los Angeles, California, USA. November 7-12, 1998. Society for Neuroscience.
ISSN: 0190-5295.
T Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
A English
D Entered STN: 10 Feb 1999
Last Updated on STN: 10 Feb 1999

3 ANSWER 6 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
N 1998:50599 BIOSIS
N PREV199800050599

TI Selective inhibition of oncogenic ras-p21 in vivo by agents that block its interaction with jun- ***N*** - ***kinase*** (JNK) and jun proteins. Implications for the design of selective chemotherapeutic agents.
AU Amar, Shazia; Glozman, Albert; Chung, Denise; Adler, Victor; Ronai, Zeev; Friedman, Fred K.; Robinson, Richard; Brandt-Rauf, Paul; Yamaizumi, Z.; Pincus, Matthew R. [Reprint author]
CS Dep. Pathol. Lab. Med., Veterans Affairs Med. Cent., 800 Poly Place, Brooklyn, NY 11209, USA
SO Cancer Chemotherapy and Pharmacology, (Dec., 1997) Vol. 41, No. 1, pp. 79-85. print.
CODEN: CCPHDZ. ISSN: 0344-5704.
DT Article
LA English
ED Entered STN: 27 Jan 1998
Last Updated on STN: 20 Mar 1998

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AN 1997:159070 BIOSIS
DN PREV199799458273
TI Conformation-dependent phosphorylation of p53.
AU Adler, Victor; Pincus, Matthew R. [Reprint author]; Minamoto, Toshinari; Fuchs, Serge Y.; Bluth, Mark J.; Brandt-Rauf, Paul W.; Friedman, Fred K.; Robinson, Richard C.; Chen, James M.; Wang, Xin Wei; Harris, Curtis C.; Ronai, Ze'ev
CS Dep. Pathol. Lab. Med., SUNY Health Sci. Cent., Brooklyn, NY 11209, USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 5, pp. 1686-1691.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 15 Apr 1997
Last Updated on STN: 2 May 1997

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DN PREV199699241729
TI Phosphorylation-dependent targeting of c-Jun ubiquitination by Jun ***N*** - ***kinase*** .
AU Fuchs, Serge Y.; Dolan, Lisa; Davis, Roger J.; Ronai, Ze'ev [Reprint author]
CS Molecular Carcinogenesis Program, American Health Foundation, One Dana Road, Valhalla, New York, NY 10595, USA
SO Oncogene, (1996) Vol. 13, No. 7, pp. 1531-1535.
CODEN: ONCNES. ISSN: 0950-9232.
DT Article
LA English
ED Entered STN: 22 Nov 1996
Last Updated on STN: 23 Nov 1996

L3 ANSWER 9 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1996:511352 BIOSIS
DN PREV199699233708
TI Effects of chemopreventive selenium compounds on Jun ***N*** - ***kinase*** activities.
AU Adler, Victor; Pincus, Matthew R.; Posner, Scott; Upadhyaya, Pramod; El-Bayoumy, Karam; Ronai, Ze'ev [Reprint author]
CS Mol. Carcinogenesis Program, American Health Foundation, Valhalla, NY 10595, USA
SO Carcinogenesis (Oxford), (1996) Vol. 17, No. 9, pp. 1849-1854.
CODEN: CRNGDP. ISSN: 0143-3334.
DT Article
LA English
ED Entered STN: 14 Nov 1996
Last Updated on STN: 14 Nov 1996

L3 ANSWER 10 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1996:367626 BIOSIS
DN PREV199699089982
TI Evidence that signal transduction by oncogenic ras-p21 protein depends on its interaction with jun kinase and jun proteins.
AU Glozman, Albert; Amar, Shaziah; Chung, Denise; Adler, Victor; Ronai, Zeev; Brandt-Rauf, Paul; Nishimura, S.; Yamaizumi, Z.; Pincus, Matthew R.

[Reprint author]
CS Dep. Pathol. Lab. Med., Veterans Affairs Med. Center, 800 Poly Plce, Brooklyn, NY 11209, USA
SO Medical Science Research, (1996) Vol. 24, No. 5, pp. 331-333.
CODEN: MSCREJ. ISSN: 0269-8951.
DT Article
LA English
ED Entered STN: 14 Aug 1996
Last Updated on STN: 15 Aug 1996

L3 ANSWER 11 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1996:257724 BIOSIS
DN PREV199698813853
TI Phosphorylation-dependent targeting of c-Jun ubiquitination by Jun ***N*** - ***kinase*** .
AU Fuchs, S. [Reprint author]; Dolan, L. [Reprint author]; Davis, R. J.; Ronai, Z. [Reprint author]
CS Mol. Carcinogenesis Program, American Health Foundation, Valhalla, NY 10595, USA
SO Proceedings of the American Association for Cancer Research Annual Meeting, (1996) Vol. 37, No. 0, pp. 530.
Meeting Info.: 87th Annual Meeting of the American Association for Cancer Research. Washington, D.C., USA. April 20-24, 1996.
ISSN: 0197-016X.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 31 May 1996
Last Updated on STN: 31 May 1996

L3 ANSWER 12 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1996:254465 BIOSIS
DN PREV199698810594
TI Complexes of ras-p21 with jun- ***N*** - ***kinase*** .
AU Adler, V. [Reprint author]; Pincus, M. R.; Polotskaya, A. [Reprint author]; Montano, X.; Brandt-Rauf, P. W.; Ronai, Z. [Reprint author]
CS Mol. Carcinogenesis Program, Am. Health Found., Valhalla, NY, USA
SO Proceedings of the American Association for Cancer Research Annual Meeting, (1996) Vol. 37, No. 0, pp. 52.
Meeting Info.: 87th Annual Meeting of the American Association for Cancer Research. Washington, D.C., USA. April 20-24, 1996.
ISSN: 0197-016X.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 31 May 1996
Last Updated on STN: 31 May 1996

L3 ANSWER 13 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1996:10250 BIOSIS
DN PREV199698582385
TI In vitro complexes of ras-p21 with jun- ***N*** - ***kinase*** and c-jun proteins.
AU Adler, Victor [Reprint author]; Pincus, Matthew R.; Brandt-Raul, Paul W.; Ronai, Ze'ev
CS Mol. Carcinogenesis Program, American Health Foundation, Valhalla, NY, USA
SO International Journal of Oncology, (1995) Vol. 7, No. SUPPL., pp. 997.
Meeting Info.: 1st World Congress on Advances in Oncology. Athens, Greece. October 22-26, 1995.
ISSN: 1019-6439.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 4 Jan 1996
Last Updated on STN: 4 Jan 1996

L3 ANSWER 14 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1995:209561 BIOSIS
DN PREV199598223861
TI NGF protects PC12 cells against ischemia by a mechanism that requires the ***N*** - ***kinase*** .

AU Boniece, I. R.; Wagner, J. A. [Reprint author]
CS Dep. Cell Biol. and Anat., Cornell Univ. Med. Coll., 1300 York Ave., New York, NY 10021, USA
SO Journal of Neuroscience Research, (1995) Vol. 40, No. 1, pp. 1-9.
CODEN: JNREDK. ISSN: 0360-4012.
DT Article
LA English
ED Entered STN: 23 May 1995
Last Updated on STN: 23 May 1995

L3 ANSWER 15 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1990:130545 BIOSIS
DN PREV199089069356; BA89:69356
TI MULTIPLE PATHWAYS OF ***N*** ***KINASE*** ACTIVATION IN PC12 CELLS.
AU ROWLAND-GAGNE E [Reprint author]; GREENE L A
CS DEPARTMENT PATHOLOGY, COLUMBIA UNIVERSITY, 630 WEST 168 STREET, NEW YORK, NY 10032, USA
SO Journal of Neurochemistry, (1990) Vol. 54, No. 2, pp. 424-433.
CODEN: JONRA9. ISSN: 0022-3042.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 13 Mar 1990
Last Updated on STN: 13 Mar 1990

L3 ANSWER 16 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1990:88223 BIOSIS
DN PREV199089047574; BA89:47574
TI CONTRIBUTIONS OF VARIOUS RAT PLASMA PEPTIDASES TO KININ HYDROLYSIS.
AU ISHIDA H [Reprint author]; SCICLI A G; CARRETERO O A
CS HYPERTENSION RES DIV, HENRY FORD HOSP, 2799 W GRAND BLVD, DETROIT, MICH 48202, USA
SO Journal of Pharmacology and Experimental Therapeutics, (1989) Vol. 251, No. 3, pp. 817-820.
CODEN: JPETAB. ISSN: 0022-3565.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 9 Feb 1990
Last Updated on STN: 9 Feb 1990

L3 ANSWER 17 OF 100 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1989:135118 BIOSIS
DN PREV198987069771; BA87:69771
TI COMPLEMENTARY DNA CLONING AND COMPLETE PRIMARY STRUCTURE OF THE SMALL ACTIVE SUBUNIT OF HUMAN CARBOXYPEPTIDASE ***N*** ***KINASE*** 1.
AU GEBHARD W [Reprint author]; SCHUBE M; EULITZ M
CS ABT FUER KLIN CHEM UND KLIN BIOCHEM IN DER CHIR KLIN INNENSTADT, UNIV MUENCHEN, NUSSBAUMSTRASSE 20, D-8000 MUENCHEN, W GER
SO European Journal of Biochemistry, (1989) Vol. 178, No. 3, pp. 603-608.
CODEN: EJBCAI. ISSN: 0014-2956.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 10 Mar 1989
Last Updated on STN: 10 Mar 1989

L3 ANSWER 18 OF 100 CANCERLIT on STN
AN 97622087 CANCERLIT
DN 97622087
TI Stress-activated signal transduction pathways in human glioma cell lines exposed to thapsigargin and 4-aminopyridine (Meeting abstract).
AU Singh S; Rami B; Chin L
CS University of Maryland Medical Systems, Baltimore, MD 21201.
SO Proc Annu Meet Am Assoc Cancer Res, ***(1997)*** 38 A945.
ISSN: 0197-016X.
DT (MEETING ABSTRACTS)
LA English
FS Institute for Cell and Developmental Biology
EM 199711
ED Entered STN: 19980417
Last Updated on STN: 19980417

L3 ANSWER 19 OF 100 CANCERLIT on STN
AN 97609811 CANCERLIT
DN 97609811
TI In vitro complexes of ras-p21 with jun- ***N*** - ***kinase*** and c-jun proteins (Meeting abstract).
AU Adler V; Pincus M R; Brandt-Raul P W; Ronai Z
CS Molecular Carcinogenesis Program, American Health Foundation, Valhalla, NY.
SO Int J Oncol, ***(1995)*** 7 (Suppl) 997.
ISSN: 1019-6439.
DT (MEETING ABSTRACTS)
LA English
FS Institute for Cell and Developmental Biology
EM 199705
ED Entered STN: 19980417
Last Updated on STN: 19980417

L3 ANSWER 20 OF 100 CANCERLIT on STN
AN 97608791 CANCERLIT
DN 97608791
TI Phosphorylation-dependent targeting of c-Jun ubiquitination by Jun ***N*** - ***kinase*** (Meeting abstract).
AU Fuchs S; Dolan L; Davis R J; Ronai Z
CS Molecular Carcinogenesis Program, American Health Foundation, Valhalla, NY 10595.
SO Proc Annu Meet Am Assoc Cancer Res, ***(1996)*** 37 A3625.
ISSN: 0197-016X.
DT (MEETING ABSTRACTS)
LA English
FS Institute for Cell and Developmental Biology
EM 199704
ED Entered STN: 19980417
Last Updated on STN: 19980417

L3 ANSWER 21 OF 100 CANCERLIT on STN
AN 96625841 CANCERLIT
DN 96625841
TI Complexes of ras-p21 with jun- ***N*** - ***kinase*** (Meeting abstract).
AU Adler V; Pincus M R; Polotskaya A; Montano X; Brandt-Rauf P W; Ronai Z
CS Molecular Carcinogenesis Program, American Health Foundation, Valhalla, NY 10595.
SO Proc Annu Meet Am Assoc Cancer Res, ***(1996)*** 37 A359.
ISSN: 0197-016X.
DT (MEETING ABSTRACTS)
LA English
FS Institute for Cell and Developmental Biology
EM 199606
ED Entered STN: 19970509
Last Updated on STN: 19970509

L3 ANSWER 22 OF 100 CANCERLIT on STN
AN 90657021 CANCERLIT
DN 90657021
TI THE CHARACTERIZATION, PARTIAL PURIFICATION AND REGULATION OF AN NGF-ACTIVATED PROTEIN KINASE IN PC12 CELLS.
AU Gagne E R
CS New York Univ., NY.
SO Diss Abstr Int [B], ***(1989)*** 49 (9) 3551.
ISSN: 0419-4217.
DT (THESIS)
LA English
FS Institute for Cell and Developmental Biology
EM 198912
ED Entered STN: 19941107
Last Updated on STN: 19970509

L3 ANSWER 23 OF 100 CANCERLIT on STN
AN 90132665 CANCERLIT
DN 90132665 PubMed ID: 2153751
TI Multiple pathways of ***N*** - ***kinase*** activation in PC12 cells.
AU Rowland-Gagne E; Greene L A
CS Department of Pharmacology, New York University School of Medicine.
NC GM 07238 (NIGMS)

NS16036 (NINDS)
SO JOURNAL OF NEUROCHEMISTRY, ***(1990 Feb)*** 54 (2) 423-33.
Journal code: 2985190R. ISSN: 0022-3042.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS MEDLINE; Priority Journals
OS MEDLINE 90132665
EM 199002
ED Entered STN: 19941107
Last Updated on STN: 19970509

L3 ANSWER 24 OF 100 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1995:902214 CAPLUS
DN 123:309119
TI Protein kinases and phosphatases that act on histidine, lysine, or arginine residues in eukaryotic proteins: a possible regulator of the mitogen-activated protein kinase cascade
AU Matthews, Harry R.
CS Department Biological Chemistry, University California Davis, Davis, 95616, USA
SO Pharmacology & Therapeutics (***1995***), 67(3), 323-50
CODEN: PHTHDT; ISSN: 0163-7258
PB Elsevier
DT Journal; General Review
LA English

L3 ANSWER 25 OF 100 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1989:434215 CAPLUS
DN 111:34215
TI The characterization, partial purification, and regulation of an NGF-activated protein kinase in PC12 cells
AU Gagne, Elizabeth Rowland
CS New York Univ., New York, NY, USA
SO (***1988***) 166 pp. Avail.: Univ. Microfilms Int., Order No. DA8825019
From: Diss. Abstr. Int. B 1989, 49(9), 3551-2
DT Dissertation
LA English

L3 ANSWER 26 OF 100 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1986:568681 CAPLUS
DN 105:168681
TI Modulation of the interaction between chemotactic cAMP-receptor and N-protein by cAMP-dependent kinase in Dictyostelium discoideum membranes
AU Luderus, M. E. E.; Van der Meer, R. F.; Van Driel, R.
CS Lab. Biochem., Univ. Amsterdam, Amsterdam, 1000 HD, Neth.
SO FEBS Letters (***1986***), 205(2), 189-94
CODEN: FEBLAL; ISSN: 0014-5793
DT Journal
LA English

L3 ANSWER 27 OF 100 DISSABS COPYRIGHT (C) 2004 ProQuest Information and Learning Company; All Rights Reserved on STN
AN 88:18581 DISSABS Order Number: AAR8825019
TI THE CHARACTERIZATION, PARTIAL PURIFICATION AND REGULATION OF AN NGF-ACTIVATED PROTEIN KINASE IN PC12 CELLS
AU GAGNE, ELIZABETH ROWLAND [PH.D.]; GREENE, LLOYD A. [advisor]
CS NEW YORK UNIVERSITY (0146)
SO Dissertation Abstracts International, (***1988***) Vol. 49, No. 9B, p. 3551. Order No.: AAR8825019. 166 pages.
DT Dissertation
FS DAI
LA English
ED Entered STN: 19921118
Last Updated on STN: 19921118

L3 ANSWER 28 OF 100 DISSABS COPYRIGHT (C) 2004 ProQuest Information and Learning Company; All Rights Reserved on STN
AN 85:11026 DISSABS Order Number: AAR8521211
TI HISTIDINE KINASE ACTIVITY IN THE NUCLEUS OF PHYSARUM POLYCEPHALUM (PROTEIN, HISTONE PHOSPHORYLATION)
AU HUEBNER, VERENA DORIS [PH.D.]
CS UNIVERSITY OF CALIFORNIA, DAVIS (0029)
SO Dissertation Abstracts International, (***1985***) Vol. 46, No. 7B, p. 2292. Order No.: AAR8521211. 151 pages.

DT Dissertation
FS DAI
LA English
ED Entered STN: 19921118
    Last Updated on STN: 19921118

L3 ANSWER 29 OF 100 GENBANK.RTM. COPYRIGHT 2004 on STN

LOCUS (LOC): BX842581 GenBank (R)
GenBank ACC. NO. (GBN): BX842581 AL008883 AL008967 AL021070 AL021287 AL021309 AL123456 Z74024 Z74697 Z81331 Z83018 Z83857 Z83858 Z83866 Z95150 Z95207
GenBank VERSION (VER): BX842581.1 GI:41352756
CAS REGISTRY NO. (RN): 644747-76-2
SEQUENCE LENGTH (SQL): 348676
MOLECULE TYPE (CI): DNA; linear
DIVISION CODE (CI): Bacteria
DATE (DATE): 10 Jun 2004
DEFINITION (DEF): Mycobacterium tuberculosis H37Rv complete genome; segment 10/13.
KEYWORDS (ST): complete genome
SOURCE: Mycobacterium tuberculosis H37Rv
 ORGANISM (ORGN): Mycobacterium tuberculosis H37Rv
Bacteria; Actinobacteria; Actinobacteridae; Actinomycetales; Corynebacterineae; Mycobacteriaceae; Mycobacterium; Mycobacterium tuberculosis complex
COMMENT:
    On or before Jan 28, 2004 this sequence version replaced gi:3261490, gi:3261491, gi:3261496, gi:3261508, gi:3261510, gi:3250700, gi:3261602, gi:3261650, gi:3261671, gi:3242252, gi:3261675, gi:3261691, gi:3261745.
    Notes:
    Details of M. tuberculosis sequencing at the Sanger Centre are available on the World Wide Web.
    (URL, http://www.sanger.ac.uk/Projects/M_tuberculosis/).
REFERENCE: 1
  AUTHOR (AU): Cole,S.T.; Brosch,R.; Parkhill,J.; Garnier,T.; Churcher,C.; Harris,D.; Gordon,S.V.; Eiglmeier,K.; Gas,S.; Barry III,C.E.; Tekaia,F.; Badcock,K.; Basham,D.; Brown,D.; Chillingworth,T.; Connor,R.; Davies,R.; Devlin,K.; Feltwell,T.; Gentles,S.; Hamlin,N.; Holroyd,S.; Hornsby,T.; Jagels,K.; Krogh,A.; McLean,J.; Moule,S.; Murphy,L.; Oliver,S.; Osborne,J.; Quail,M.A.; Rajandream,M.A.; Rogers,J.; Rutter,S.; Seeger,K.; Skelton,S.; Squares,S.; Squares,R.; Sulston,J.E.; Taylor,K.; Whitehead,S.; Barrell,B.G.
  TITLE (TI): Deciphering the biology of Mycobacterium tuberculosis from the complete genome sequence
  JOURNAL (SO): Nature, 393 (6685), 537-544 (***1998***)
REFERENCE: 2
  AUTHOR (AU): Camus,J.C.; Pryor,M.J.; Medigue,C.; Cole,S.T.
  TITLE (TI): Re-annotation of the genome sequence of Mycobacterium tuberculosis H37Rv
  JOURNAL (SO): Microbiology (Reading, Engl.), 148 (Pt 10), 2967-2973 (2002)
REFERENCE: 3 (bases 1 to 348676)
  AUTHOR (AU): Parkhill,J.
  TITLE (TI): Direct Submission
  JOURNAL (SO): Submitted (11-JUN-1998) Submitted on behalf of the Mycobacterium tuberculosis sequencing and mapping teams, Sanger Centre, Wellcome Trust Genome Campus, Hinxton, Cambridge CB10 1SA Unite de Genetique Moleculaire Bacterienne, Institut Pasteur, 28 rue du Docteur Roux, 75724 Paris Cedex 15, France E-mail: parkhill@sanger.ac.uk

=> S L3 AND phosphorylation
  41 FILES SEARCHED...
L4 37 L3 AND PHOSPHORYLATION

=> D L4 1-37

L4 ANSWER 1 OF 37 AGRICOLA Compiled and distributed by the National Agricultural Library of the Department of Agriculture of the United States of America. It contains copyrighted materials. All rights reserved. (2004) on STN

AN 93:11119 AGRICOLA
DN IND93000597
TI N-terminal mutations modulate yeast SNF1 protein kinase function.
AU Estruch, F.; Treitel, M.A.; Yang, X.L.; Carlson, M.
CS Columbia University, New York, NY
AV DNAL (442.8 G28)
SO Genetics, ***Nov 1992.*** Vol. 132, No. 3. p. 639-650
Publisher: Baltimore, Md. : Genetics Society of America.
CODEN: GENTAE; ISSN: 0016-6731
NTE Includes references.
DT Article
FS U.S. Imprints not USDA, Experiment or Extension
LA English

L4 ANSWER 2 OF 37 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2002:251976 BIOSIS
DN PREV200200251976
TI Genetic mechanisms and MAPK-signaling in post-infarction heart failure in rats.
AU Gurevich, Andrey K. [Reprint author]; Weinberger, Howard D. [Reprint author]; Nemenoff, Raphael A. [Reprint author]; Bedigian, Martin P.; Schrier, Robert W. [Reprint author]
CS Department of Medicine, University of Colorado Health Sciences Center, Denver, CO, USA
SO Journal of the American Society of Nephrology, (September, 2000) Vol. 11, No. Program and Abstract Issue, pp. 455A. print.
Meeting Info.: 33rd Annual Meeting of the American Society of Nephrology and the 2000 Renal Week. Toronto, Ontario, Canada. October 10-16, 2000. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 24 Apr 2002
Last Updated on STN: 24 Apr 2002

L4 ANSWER 3 OF 37 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1997:159070 BIOSIS
DN PREV199799458273
TI Conformation-dependent ***phosphorylation*** of p53.
AU Adler, Victor; Pincus, Matthew R. [Reprint author]; Minamoto, Toshinari; Fuchs, Serge Y.; Bluth, Mark J.; Brandt-Rauf, Paul W.; Friedman, Fred K.; Robinson, Richard C.; Chen, James M.; Wang, Xin Wei; Harris, Curtis C.; Ronai, Ze'ev
CS Dep. Pathol. Lab. Med., SUNY Health Sci. Cent., Brooklyn, NY 11209, USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 5, pp. 1686-1691.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 15 Apr 1997
Last Updated on STN: 2 May 1997

L4 ANSWER 4 OF 37 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1996:519373 BIOSIS
DN PREV199699241729
TI ***Phosphorylation*** -dependent targeting of c-Jun ubiquitination by Jun ***N*** - ***kinase*** .
AU Fuchs, Serge Y.; Dolan, Lisa; Davis, Roger J.; Ronai, Ze'ev [Reprint author]
CS Molecular Carcinogenesis Program, American Health Foundation, One Dana Road, Valhalla, New York, NY 10595, USA
SO Oncogene, (1996) Vol. 13, No. 7, pp. 1531-1535.
CODEN: ONCNES. ISSN: 0950-9232.
DT Article
LA English
ED Entered STN: 22 Nov 1996
Last Updated on STN: 23 Nov 1996

L4 ANSWER 5 OF 37 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1996:257724 BIOSIS

DN PREV199698813853
TI ***Phosphorylation*** -dependent targeting of c-Jun ubiquitination by Jun ***N*** - ***kinase*** .
AU Fuchs, S. [Reprint author]; Dolan, L. [Reprint author]; Davis, R. J.; Ronai, Z. [Reprint author]
CS Mol. Carcinogenesis Program, American Health Foundation, Valhalla, NY 10595, USA
SO Proceedings of the American Association for Cancer Research Annual Meeting, (1996) Vol. 37, No. 0, pp. 530.
Meeting Info.: 87th Annual Meeting of the American Association for Cancer Research. Washington, D.C., USA. April 20-24, 1996.
ISSN: 0197-016X.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 31 May 1996
Last Updated on STN: 31 May 1996

L4 ANSWER 6 OF 37 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1996:254465 BIOSIS
DN PREV199698810594
TI Complexes of ras-p21 with jun- ***N*** - ***kinase*** .
AU Adler, V. [Reprint author]; Pincus, M. R.; Polotskaya, A. [Reprint author]; Montano, X.; Brandt-Rauf, P. W.; Ronai, Z. [Reprint author]
CS Mol. Carcinogenesis Program, Am. Health Found., Valhalla, NY, USA
SO Proceedings of the American Association for Cancer Research Annual Meeting, (1996) Vol. 37, No. 0, pp. 52.
Meeting Info.: 87th Annual Meeting of the American Association for Cancer Research. Washington, D.C., USA. April 20-24, 1996.
ISSN: 0197-016X.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 31 May 1996
Last Updated on STN: 31 May 1996

L4 ANSWER 7 OF 37 CANCERLIT on STN
AN 97609811 CANCERLIT
DN 97609811
TI In vitro complexes of ras-p21 with jun- ***N*** - ***kinase*** and c-jun proteins (Meeting abstract).
AU Adler V; Pincus M R; Brandt-Raul P W; Ronai Z
CS Molecular Carcinogenesis Program, American Health Foundation, Valhalla, NY.
SO Int J Oncol, ***(1995)*** 7 (Suppl) 997.
ISSN: 1019-6439.
DT (MEETING ABSTRACTS)
LA English
FS Institute for Cell and Developmental Biology
EM 199705
ED Entered STN: 19980417
Last Updated on STN: 19980417

L4 ANSWER 8 OF 37 CANCERLIT on STN
AN 97608791 CANCERLIT
DN 97608791
TI ***Phosphorylation*** -dependent targeting of c-Jun ubiquitination by Jun ***N*** - ***kinase*** (Meeting abstract).
AU Fuchs S; Dolan L; Davis R J; Ronai Z
CS Molecular Carcinogenesis Program, American Health Foundation, Valhalla, NY 10595.
SO Proc Annu Meet Am Assoc Cancer Res, ***(1996)*** 37 A3625.
ISSN: 0197-016X.
DT (MEETING ABSTRACTS)
LA English
FS Institute for Cell and Developmental Biology
EM 199704
ED Entered STN: 19980417
Last Updated on STN: 19980417

L4 ANSWER 9 OF 37 CANCERLIT on STN
AN 96625841 CANCERLIT
DN 96625841
TI Complexes of ras-p21 with jun- ***N*** - ***kinase*** (Meeting

abstract).
AU Adler V; Pincus M R; Polotskaya A; Montano X; Brandt-Rauf P W; Ronai Z
CS Molecular Carcinogenesis Program, American Health Foundation, Valhalla, NY 10595.
SO Proc Annu Meet Am Assoc Cancer Res, ***(1996)*** 37 A359.
ISSN: 0197-016X.
DT (MEETING ABSTRACTS)
LA English
FS Institute for Cell and Developmental Biology
EM 199606
ED Entered STN: 19970509
Last Updated on STN: 19970509

L4 ANSWER 10 OF 37 CANCERLIT on STN
AN 90657021 CANCERLIT
DN 90657021
TI THE CHARACTERIZATION, PARTIAL PURIFICATION AND REGULATION OF AN NGF-ACTIVATED PROTEIN KINASE IN PC12 CELLS.
AU Gagne E R
CS New York Univ., NY.
SO Diss Abstr Int [B], ***(1989)*** 49 (9) 3551.
ISSN: 0419-4217.
DT (THESIS)
LA English
FS Institute for Cell and Developmental Biology
EM 198912
ED Entered STN: 19941107
Last Updated on STN: 19970509

L4 ANSWER 11 OF 37 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1995:902214 CAPLUS
DN 123:309119
TI Protein kinases and phosphatases that act on histidine, lysine, or arginine residues in eukaryotic proteins: a possible regulator of the mitogen-activated protein kinase cascade
AU Matthews, Harry R.
CS Department Biological Chemistry, University California Davis, Davis, 95616, USA
SO Pharmacology & Therapeutics (***1995***), 67(3), 323-50
CODEN: PHTHDT; ISSN: 0163-7258
PB Elsevier
DT Journal; General Review
LA English

L4 ANSWER 12 OF 37 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1986:568681 CAPLUS
DN 105:168681
TI Modulation of the interaction between chemotactic cAMP-receptor and N-protein by cAMP-dependent kinase in Dictyostelium discoideum membranes
AU Luderus, M. E. E.; Van der Meer, R. F.; Van Driel, R.
CS Lab. Biochem., Univ. Amsterdam, Amsterdam, 1000 HD, Neth.
SO FEBS Letters (***1986***), 205(2), 189-94
CODEN: FEBLAL; ISSN: 0014-5793
DT Journal
LA English

L4 ANSWER 13 OF 37 DISSABS COPYRIGHT (C) 2004 ProQuest Information and Learning Company; All Rights Reserved on STN
AN 88:18581 DISSABS Order Number: AAR8825019
TI THE CHARACTERIZATION, PARTIAL PURIFICATION AND REGULATION OF AN NGF-ACTIVATED PROTEIN KINASE IN PC12 CELLS
AU GAGNE, ELIZABETH ROWLAND [PH.D.]; GREENE, LLOYD A. [advisor]
CS NEW YORK UNIVERSITY (0146)
SO Dissertation Abstracts International, (***1988***) Vol. 49, No. 9B, p. 3551. Order No.: AAR8825019. 166 pages.
DT Dissertation
FS DAI
LA English
ED Entered STN: 19921118
Last Updated on STN: 19921118

L4 ANSWER 14 OF 37 DISSABS COPYRIGHT (C) 2004 ProQuest Information and Learning Company; All Rights Reserved on STN
AN 85:11026 DISSABS Order Number: AAR8521211
TI HISTIDINE KINASE ACTIVITY IN THE NUCLEUS OF PHYSARUM POLYCEPHALUM (PROTEIN, HISTONE ***PHOSPHORYLATION***)

AU HUEBNER, VERENA DORIS [PH.D.]
CS UNIVERSITY OF CALIFORNIA, DAVIS (0029)
SO Dissertation Abstracts International, (***1985***) Vol. 46, No. 7B, p. 2292. Order No.: AAR8521211. 151 pages.
DT Dissertation
FS DAI
LA English
ED Entered STN: 19921118
Last Updated on STN: 19921118

L4 ANSWER 15 OF 37 GENBANK.RTM. COPYRIGHT 2004 on STN

LOCUS (LOC): BX842581 GenBank (R)
GenBank ACC. NO. (GBN): BX842581 AL008883 AL008967 AL021070 AL021287 AL021309 AL123456 Z74024 Z74697 Z81331 Z83018 Z83857 Z83858 Z83866 Z95150 Z95207
GenBank VERSION (VER): BX842581.1 GI:41352756
CAS REGISTRY NO. (RN): 644747-76-2
SEQUENCE LENGTH (SQL): 348676
MOLECULE TYPE (CI): DNA; linear
DIVISION CODE (CI): Bacteria
DATE (DATE): 10 Jun 2004
DEFINITION (DEF): Mycobacterium tuberculosis H37Rv complete genome; segment 10/13.
KEYWORDS (ST): complete genome
SOURCE: Mycobacterium tuberculosis H37Rv
ORGANISM (ORGN): Mycobacterium tuberculosis H37Rv
Bacteria; Actinobacteria; Actinobacteridae; Actinomycetales; Corynebacterineae; Mycobacteriaceae; Mycobacterium; Mycobacterium tuberculosis complex
COMMENT:
On or before Jan 28, 2004 this sequence version replaced gi:3261490, gi:3261491, gi:3261496, gi:3261508, gi:3261510, gi:3250700, gi:3261602, gi:3261650, gi:3261671, gi:3242252, gi:3261675, gi:3261691, gi:3261745.
Notes:
Details of M. tuberculosis sequencing at the Sanger Centre are available on the World Wide Web.
(URL, http://www.sanger.ac.uk/Projects/M_tuberculosis/).
REFERENCE: 1
AUTHOR (AU): Cole,S.T.; Brosch,R.; Parkhill,J.; Garnier,T.; Churcher,C.; Harris,D.; Gordon,S.V.; Eiglmeier,K.; Gas,S.; Barry III,C.E.; Tekaia,F.; Badcock,K.; Basham,D.; Brown,D.; Chillingworth,T.; Connor,R.; Davies,R.; Devlin,K.; Feltwell,T.; Gentles,S.; Hamlin,N.; Holroyd,S.; Hornsby,T.; Jagels,K.; Krogh,A.; McLean,J.; Moule,S.; Murphy,L.; Oliver,S.; Osborne,J.; Quail,M.A.; Rajandream,M.A.; Rogers,J.; Rutter,S.; Seeger,K.; Skelton,S.; Squares,S.; Sqares,R.; Sulston,J.E.; Taylor,K.; Whitehead,S.; Barrell,B.G.
TITLE (TI): Deciphering the biology of Mycobacterium tuberculosis from the complete genome sequence
JOURNAL (SO): Nature, 393 (6685), 537-544 (***1998***)
REFERENCE: 2
AUTHOR (AU): Camus,J.C.; Pryor,M.J.; Medigue,C.; Cole,S.T.
TITLE (TI): Re-annotation of the genome sequence of Mycobacterium tuberculosis H37Rv
JOURNAL (SO): Microbiology (Reading, Engl.), 148 (Pt 10), 2967-2973 (2002)
REFERENCE: 3 (bases 1 to 348676)
AUTHOR (AU): Parkhill,J.
TITLE (TI): Direct Submission
JOURNAL (SO): Submitted (11-JUN-1998) Submitted on behalf of the Mycobacterium tuberculosis sequencing and mapping teams, Sanger Centre, Wellcome Trust Genome Campus, Hinxton, Cambridge CB10 1SA Unite de Genetique Moleculaire Bacterienne, Institut Pasteur, 28 rue du Docteur Roux, 75724 Paris Cedex 15, France E-mail: parkhill@sanger.ac.uk

=> S N-kinase dependent phosphorylation
12 FILES SEARCHED...
24 FILES SEARCHED...
45 FILES SEARCHED...
66 FILES SEARCHED...

```
L5          20 N-KINASE DEPENDENT PHOSPHORYLATION

=> DUP REM L5
DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, ADISNEWS, BIOCOMMERCE, DGENE,
DRUGMONOG2, FEDRIP, FOREGE, GENBANK, IMSPRODUCT, IMSRESEARCH, KOSMET,
MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, PROUSDDR, RDISCLOSURE, SYNTHLINE'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L5
L6           17 DUP REM L5 (3 DUPLICATES REMOVED)

=> D L6 1-17

L6    ANSWER 1 OF 17  BIOTECHDS COPYRIGHT 2004 THE THOMSON CORP. on STN
      DUPLICATE 1
AN    2003-13120  BIOTECHDS
TI    Producing neurosalutary effect, and treating neurological disorder, in a
      subject, by administering a therapeutically effective amount of a
      compound that modulates the activity of N-kinase, to the subject;
         neurosalutary effect and enzyme protein modulation for use in disease
         therapy
AU    BENOWITZ L I
PA    CHILDRENS MEDICAL CENT
PI    US 2002160933 31 Oct 2002
AI    US 2001-949200 7 Sep 2001
PRAI  US 2001-949200 7 Sep 2001; US 2000-656915 7 Sep 2000
DT    Patent
LA    English
OS    WPI: 2003-328371 [31]

L6   ANSWER 2 OF 17  WPIDS  COPYRIGHT 2004 THE THOMSON CORP on STN
AN   2002-393816 [42]   WPIDS
CR   2003-328371 [31]
DNC  C2002-110736
TI   Producing a neurosalutary effect in a subject e.g., one suffering from
     neurological disorder such as stroke, to treat the subject, by
     administering a compound that modulates activity of N-kinase .
DC   B04 D16
IN   BENOWITZ, L I
PA   (CHIL-N) CHILDRENS MEDICAL CENT
CYC  97
PI   WO 2002020056   A2 20020314 (200242)* EN   42    A61K045-00
        RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
            NL OA PT SD SE SL SZ TR TZ UG ZW
         W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
            DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
            KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH PL PT RO
            RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
     AU 2001087118   A  20020322 (200251)            A61K045-00
     EP 1315514      A2 20030604 (200337)  EN        A61K038-18
         R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
            RO SE SI TR
     JP 2004523470   W  20040805 (200451)       77   A61K045-00
ADT  WO 2002020056 A2 WO 2001-US27691 20010907; AU 2001087118 A AU 2001-87118
     20010907; EP 1315514 A2 EP 2001-966619 20010907, WO 2001-US27691 20010907;
     JP 2004523470 W WO 2001-US27691 20010907, JP 2002-524539 20010907
FDT  AU 2001087118 A Based on WO 2002020056; EP 1315514 A2 Based on WO
     2002020056; JP 2004523470 W Based on WO 2002020056
PRAI US 2000-656915       20000907
IC   ICM  A61K038-18; A61K045-00
     ICS  A61K009-10; A61K009-127; A61P009-10; A61P009-12; A61P025-00;
          A61P025-02; A61P025-08; A61P025-14; A61P025-16; A61P025-18;
          A61P025-24; A61P025-28; A61P043-00; C07K014-475; C07K016-40;
          C12N009-12; C12N015-09; C12Q001-48; G01N033-15; G01N033-50;
          G01N033-53; G01N033-566

L6   ANSWER 3 OF 17       GENBANK.RTM.  COPYRIGHT 2004 on STN

LOCUS (LOC):            BX571966     GenBank (R)
GenBank ACC. NO. (GBN): BX571966
GenBank VERSION (VER):  BX571966.1  GI:52211453
CAS REGISTRY NO. (RN):  755924-53-9
SEQUENCE LENGTH (SQL):  3173005
MOLECULE TYPE (CI):     DNA; circular
DIVISION CODE (CI):     Bacteria
DATE (DATE):            16 Sep 2004
DEFINITION (DEF):       Burkholderia pseudomallei strain K96243, chromosome 2,
```

complete sequence.
SOURCE: Burkholderia pseudomallei K96243
ORGANISM (ORGN): Burkholderia pseudomallei K96243
Bacteria; Proteobacteria; Betaproteobacteria;
Burkholderiales; Burkholderiaceae; Burkholderia;
pseudomallei group
REFERENCE: 1 (bases 1 to 3173005)
AUTHOR (AU): Holden,M.T.G.; Titball,R.W.; Peacock,S.J.;
Cerdeno-Tarraga,A.M.; Atkins,T.; Crossman,L.C.;
Pitt,T.; Churcher,C.; Mungall,K.; Bentley,S.D.;
Sebaihia,M.; Thomson,N.R.; Bason,N.; Beacham,I.R.;
Brooks,K.; Brown,K.A.; Brown,N.F.; Challis,G.L.;
Cherevach,I.; Chillingworth,T.; Cronin,A.; Crosset,B.;
Davis,P.; DeShazer,D.; Feltwell,T.; Fraser,A.;
Hance,Z.; Hauser,H.; Holroyd,S.; Jagels,K.; Keith,K.E.;
Maddison,M.; Moule,S.; Price,C.; Quail,M.A.;
Rabbinowitsch,E.; Rutherford,K.; Sanders,M.;
Simmonds,M.; Songsivilai,S.; Stevens,K.; Tumapa,S.;
Vesaratchavest,M.; Whitehead,S.; Yeats,C.;
Barrell,B.G.; Oyston,P.C.F.; Parkhill,J.
TITLE (TI): Genomic plasticity of the causative agent of
melioidosis, Burkholderia pseudomallei
JOURNAL (SO): Unpublished
REFERENCE: 2 (bases 1 to 3173005)
AUTHOR (AU): Holden,M.T.G.
TITLE (TI): Direct Submission
JOURNAL (SO): Submitted (01-SEP-2004) Submitted on behalf of the
Pathogen Sequencing Unit, Sanger Institute, Wellcome
Trust Genome Campus, Hinxton, Cambridge CB10 1SA,
E-mail: mh3@sanger.ac.uk

FEATURES (FEAT):

Feature Key	Location	Qualifier
source	1..3173005	/organism="Burkholderia pseudomallei K96243" /mol-type="genomic DNA" /strain="K96243" /db-xref="taxon:272560" /chromosome="2"

L6 ANSWER 4 OF 17 GENBANK.RTM. COPYRIGHT 2004 on STN

LOCUS (LOC): BX571965 GenBank (R)
GenBank ACC. NO. (GBN): BX571965
GenBank VERSION (VER): BX571965.1 GI:52208053
CAS REGISTRY NO. (RN): 755924-52-8
SEQUENCE LENGTH (SQL): 4074542
MOLECULE TYPE (CI): DNA; circular
DIVISION CODE (CI): Bacteria
DATE (DATE): 16 Sep 2004
DEFINITION (DEF): Burkholderia pseudomallei strain K96243, chromosome 1,
complete sequence.
SOURCE: Burkholderia pseudomallei K96243
ORGANISM (ORGN): Burkholderia pseudomallei K96243
Bacteria; Proteobacteria; Betaproteobacteria;
Burkholderiales; Burkholderiaceae; Burkholderia;
pseudomallei group
REFERENCE: 1 (bases 1 to 4074542)
AUTHOR (AU): Holden,M.T.G.; Titball,R.W.; Peacock,S.J.;
Cerdeno-Tarraga,A.M.; Atkins,T.; Crossman,L.C.;
Pitt,T.; Churcher,C.; Mungall,K.; Bentley,S.D.;
Sebaihia,M.; Thomson,N.R.; Bason,N.; Beacham,I.R.;
Brooks,K.; Brown,K.A.; Brown,N.F.; Challis,G.L.;
Cherevach,I.; Chillingworth,T.; Cronin,A.; Crosset,B.;
Davis,P.; DeShazer,D.; Feltwell,T.; Fraser,A.;
Hance,Z.; Hauser,H.; Holroyd,S.; Jagels,K.; Keith,K.E.;
Maddison,M.; Moule,S.; Price,C.; Quail,M.A.;
Rabbinowitsch,E.; Rutherford,K.; Sanders,M.;
Simmonds,M.; Songsivilai,S.; Stevens,K.; Tumapa,S.;
Vesaratchavest,M.; Whitehead,S.; Yeats,C.;
Barrell,B.G.; Oyston,P.C.F.; Parkhill,J.
TITLE (TI): Genomic plasticity of the causative agent of
melioidosis, Burkholderia pseudomallei
JOURNAL (SO): Unpublished

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REFERENCE:              2  (bases 1 to 4074542)
   AUTHOR (AU):         Holden,M.T.G.
   TITLE (TI):          Direct Submission
   JOURNAL (SO):        Submitted (01-SEP-2004) Submitted on behalf of the
                        Pathogen Sequencing Unit, Sanger Institute, Wellcome
                        Trust Genome Campus, Hinxton, Cambridge CB10 1SA,
                        E-mail: mh3@sanger.ac.uk

FEATURES (FEAT):
  Feature Key       Location                Qualifier
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source          1..4074542              /organism="Burkholderia
                                        pseudomallei K96243"
                                        /mol-type="genomic DNA"
                                        /strain="K96243"
                                        /db-xref="taxon:272560"
                                        /chromosome="1"


L6   ANSWER 5 OF 17       GENBANK.RTM.  COPYRIGHT 2004 on STN

LOCUS (LOC):            CP000002     GenBank (R)
GenBank ACC. NO. (GBN): CP000002
GenBank VERSION (VER):  CP000002.1  GI:52001702
CAS REGISTRY NO. (RN):  746492-99-9
SEQUENCE LENGTH (SQL):  4222336
MOLECULE TYPE (CI):     DNA; circular
DIVISION CODE (CI):     Bacteria
DATE (DATE):            12 Oct 2004
DEFINITION (DEF):       Bacillus licheniformis DSM 13, complete genome.
SOURCE:                 Bacillus licheniformis DSM 13
 ORGANISM (ORGN):       Bacillus licheniformis DSM 13
                        Bacteria; Firmicutes; Bacillales; Bacillaceae; Bacillus
REFERENCE:              1  (bases 1 to 4222336)
   AUTHOR (AU):         Rey,M.W.; Ramaiya,P.; Nelson,B.A.; Brody-Karpin,S.D.;
                        Zaretsky,E.J.; Tang,M.; de Leon,A.L.; Xiang,H.;
                        Gusti,V.; Clausen,I.G.; Olsen,P.B.; Rasmussen,M.D.;
                        Andersen,J.T.; Jorgensen,P.L.; Larsen,T.S.; Sorokin,A.;
                        Bolotin,A.; Lapidus,A.; Galleron,N.; Ehrlich,S.D.;
                        Berka,R.M.
   TITLE (TI):          Complete genome sequence of the industrial bacterium
                        Bacillus licheniformis and comparisons with closely
                        related Bacillus species
   JOURNAL (SO):        Genome Biol., 5, R77 (2004)
REFERENCE:              2  (bases 1 to 4222336)
   AUTHOR (AU):         Berka,R.M.; Rey,M.W.; Ramaiya,P.
   TITLE (TI):          Direct Submission
   JOURNAL (SO):        Submitted (14-JUL-2004) Novozymes Biotech Inc, 1445
                        Drew Ave, Davis, CA 95616, USA

FEATURES (FEAT):
  Feature Key       Location                Qualifier
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source          1..4222336              /organism="Bacillus licheniformis
                                        DSM 13"
                                        /mol-type="genomic DNA"
                                        /strain="ATCC 14580"
                                        /db-xref="ATCC:14580"
                                        /db-xref="taxon:279010"


L6   ANSWER 6 OF 17       GENBANK.RTM.  COPYRIGHT 2004 on STN

LOCUS (LOC):            BX950851     GenBank (R)
GenBank ACC. NO. (GBN): BX950851
GenBank VERSION (VER):  BX950851.1  GI:49609491
CAS REGISTRY NO. (RN):  726687-36-1
SEQUENCE LENGTH (SQL):  5064019
MOLECULE TYPE (CI):     DNA; circular
DIVISION CODE (CI):     Bacteria
DATE (DATE):            1 Jul 2004
DEFINITION (DEF):       Erwinia carotovora subsp. atroseptica SCRI1043,
                        complete genome.
KEYWORDS (ST):          complete genome
SOURCE:                 Erwinia carotovora subsp. atroseptica SCRI1043
 ORGANISM (ORGN):       Erwinia carotovora subsp. atroseptica SCRI1043
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Bacteria; Proteobacteria; Gammaproteobacteria;
Enterobacteriales; Enterobacteriaceae; Pectobacterium
REFERENCE: 1 (bases 1 to 5064019)
AUTHOR (AU): Bell,K.S.; Sebaihia,M.; Pritchard,L.; Holden,M.; Hyman,L.J.; Holeva,M.C.; Thomson,N.R.; Bentley,S.D.; Churcher,C.; Mungall,K.; Atkin,R.; Bason,N.; Brooks,K.; Chillingworth,T.; Clark,K.; Doggett,J.; Fraser,A.; Hance,Z.; Hauser,H.; Jagels,K.; Moule,S.; Norbertczak,H.; Ormond,D.; Price,C.; Quail,M.A.; Sanders,M.; Walker,D.; Whitehead,S.; Salmond,G.P.C.; Birch,P.R.J.; Barrell,B.G.; Parkhill,J.; Toth,I.K.
TITLE (TI): The genome sequence of the enterobacterial phytopathogen Erwinia carotovora subsp. atroseptica SCRI1043 and functional genomic identification of novel virulence factors
JOURNAL (SO): Unpublished
REFERENCE: 2 (bases 1 to 5064019)
AUTHOR (AU): Sebaihia,M.
TITLE (TI): Direct Submission
JOURNAL (SO): Submitted (18-FEB-2004) Submitted on behalf of the Pathogen Sequencing Unit, Sanger Institute, Wellcome Trust Genome Campus, Hinxton, Cambridge CB10 1SA E-mail: ms5@sanger.ac.uk

FEATURES (FEAT):

Feature Key	Location	Qualifier
source	1..5064019	/organism="Erwinia carotovora subsp. atroseptica SCRI1043" /mol-type="genomic DNA" /strain="SCRI1043" /db-xref="taxon:218491"

L6 ANSWER 7 OF 17 GENBANK.RTM. COPYRIGHT 2004 on STN

LOCUS (LOC): BX571857 GenBank (R)
GenBank ACC. NO. (GBN): BX571857
GenBank VERSION (VER): BX571857.1 GI:49243355
CAS REGISTRY NO. (RN): 726687-35-0
SEQUENCE LENGTH (SQL): 2799802
MOLECULE TYPE (CI): DNA; circular
DIVISION CODE (CI): Bacteria
DATE (DATE): 24 Jun 2004
DEFINITION (DEF): Staphylococcus aureus strain MSSA476, complete genome.
KEYWORDS (ST): complete genome
SOURCE: Staphylococcus aureus subsp. aureus MSSA476
ORGANISM (ORGN): Staphylococcus aureus subsp. aureus MSSA476
Bacteria; Firmicutes; Bacillales; Staphylococcus
REFERENCE: 1 (bases 1 to 2799802)
AUTHOR (AU): Holden,M.T.G.; Feil,E.J.; Lindsay,J.A.; Peacock,S.J.; Day,N.P.J.; Enright,M.C.; Foster,T.J.; Moore,C.E.; Hurst,L.; Atkin,R.; Barron,A.; Bason,N.; Bentley,S.D.; Chillingworth,C.; Chillingworth,T.; Churcher,C.; Clark,L.; Corton,C.; Cronin,A.; Doggett,J.; Dowd,L.; Feltwell,T.; Hance,Z.; Harris,B.; Hauser,H.; Holroyd,S.; Jagels,K.; James,K.D.; Lennard,N.; Line,A.; Mayes,R.; Moule,S.; Mungall,K.; Ormond,D.; Quail,M.A.; Rabbinowitsch,E.; Rutherford,K.; Sanders,M.; Sharp,S.; Simmonds,M.; Stevens,K.; Whitehead,S.; Barrell,B.G.; Spratt,B.G.; Parkhill,J.
TITLE (TI): Complete genomes of two clinical Staphylococcus aureus strains: evidence for the rapid evolution of virulence and drug resistance
JOURNAL (SO): Proc. Natl. Acad. Sci. U.S.A., 101 (26), 9786-9791 (2004)
OTHER SOURCE (OS): CA 141:152000
REFERENCE: 2 (bases 1 to 2799802)
AUTHOR (AU): Holden,M.T.G.
TITLE (TI): Direct Submission
JOURNAL (SO): Submitted (23-JUN-2004) Submitted on behalf of the Pathogen Sequencing Unit, Sanger Institute, Wellcome Trust Genome Campus, Hinxton, Cambridge CB10 1SA, E-mail: mh3@sanger.ac.uk

FEATURES (FEAT):

Feature Key	Location	Qualifier
source	1..2799802	/organism="Staphylococcus aureus subsp. aureus MSSA476" /mol-type="genomic DNA" /strain="MSSA476" /db-xref="taxon:282459"

L6 ANSWER 8 OF 17 GENBANK.RTM. COPYRIGHT 2004 on STN

LOCUS (LOC): BX571856 GenBank (R)
GenBank ACC. NO. (GBN): BX571856
GenBank VERSION (VER): BX571856.1 GI:49240382
CAS REGISTRY NO. (RN): 726687-34-9
SEQUENCE LENGTH (SQL): 2902619
MOLECULE TYPE (CI): DNA; circular
DIVISION CODE (CI): Bacteria
DATE (DATE): 23 Jun 2004
DEFINITION (DEF): Staphylococcus aureus subsp. aureus strain MRSA252, complete genome.
KEYWORDS (ST): complete genome
SOURCE: Staphylococcus aureus subsp. aureus MRSA252
ORGANISM (ORGN): Staphylococcus aureus subsp. aureus MRSA252
Bacteria; Firmicutes; Bacillales; Staphylococcus
REFERENCE: 1 (bases 1 to 2902619)
AUTHOR (AU): Holden,M.T.G.; Feil,E.J.; Lindsay,J.A.; Peacock,S.J.; Day,N.P.J.; Enright,M.C.; Foster,T.J.; Moore,C.E.; Hurst,L.; Atkin,R.; Barron,A.; Bason,N.; Bentley,S.D.; Chillingworth,C.; Chillingworth,T.; Churcher,C.; Clark,L.; Corton,C.; Cronin,A.; Doggett,J.; Dowd,L.; Feltwell,T.; Hance,Z.; Harris,B.; Hauser,H.; Holroyd,S.; Jagels,K.; James,K.D.; Lennard,N.; Line,A.; Mayes,R.; Moule,S.; Mungall,K.; Ormond,D.; Quail,M.A.; Rabbinowitsch,E.; Rutherford,K.; Sanders,M.; Sharp,S.; Simmonds,M.; Stevens,K.; Whitehead,S.; Barrell,B.G.; Spratt,B.G.; Parkhill,J.
TITLE (TI): Complete genomes of two clinical Staphylococcus aureus strains: evidence for the rapid evolution of virulence and drug resistance
JOURNAL (SO): Proc. Natl. Acad. Sci. U.S.A., 101 (26), 9786-9791 (2004)
OTHER SOURCE (OS): CA 141:152000
REFERENCE: 2 (bases 1 to 2902619)
AUTHOR (AU): Holden,M.T.G.
TITLE (TI): Direct Submission
JOURNAL (SO): Submitted (23-JUN-2004) Submitted on behalf of the Pathogen Sequencing Unit, Sanger Institute, Wellcome Trust Genome Campus, Hinxton, Cambridge CB10 1SA, E-mail: mh3@sanger.ac.uk

FEATURES (FEAT):

Feature Key	Location	Qualifier
source	1..2902619	/organism="Staphylococcus aureus subsp. aureus MRSA252" /mol-type="genomic DNA" /strain="MRSA252" /db-xref="taxon:282458"

L6 ANSWER 9 OF 17 GENBANK.RTM. COPYRIGHT 2004 on STN

LOCUS (LOC): BX248360 GenBank (R)
GenBank ACC. NO. (GBN): BX248360 BX248353
GenBank VERSION (VER): BX248360.1 GI:38200856
CAS REGISTRY NO. (RN): 615232-20-7
SEQUENCE LENGTH (SQL): 349659
MOLECULE TYPE (CI): DNA; linear
DIVISION CODE (CI): Bacteria
DATE (DATE): 6 Nov 2003
DEFINITION (DEF): Corynebacterium diphtheriae gravis NCTC13129, complete genome; segment 7/8.
KEYWORDS (ST): complete genome
SOURCE: Corynebacterium diphtheriae
ORGANISM (ORGN): Corynebacterium diphtheriae

Bacteria; Actinobacteria; Actinobacteridae;
Actinomycetales; Corynebacterineae; Corynebacteriaceae;
Corynebacterium

REFERENCE: 1 (bases 1 to 349659)
AUTHOR (AU): Cerdeno-Tarraga,A.M.; Efstratiou,A.; Dover,L.G.;
Holden,M.T.G.; Pallen,M.; Bentley,S.D.; Besra,G.S.;
Churcher,C.; James,K.D.; De Zoysa,A.; Chillingworth,T.;
Cronin,A.; Dowd,L.; Feltwell,T.; Hamlin,N.; Holroyd,S.;
Jagels,K.; Moule,S.; Quail,M.A.; Rabbinowitsch,E.;
Rutherford,K.; Thomson,N.R.; Unwin,L.; Whitehead,S.;
Barrell B.G. Parkhill,J.
TITLE (TI): The complete genome sequence and analysis of
Corynebacterium diphtheriae NCTC13129
JOURNAL (SO): Nucleic Acids Res., 31 (22), 6516-6523 (2003)
OTHER SOURCE (OS): CA 139:376020

REFERENCE: 2 (bases 1 to 349659)
AUTHOR (AU): Cerdeno-Tarraga,A.M.
TITLE (TI): Direct Submission
JOURNAL (SO): Submitted (03-OCT-2003) Cerdeno-Tarraga A.M., submitted
on behalf of the Pathogen Sequencing Unit, Sanger
Institute, Wellcome Trust Genome Campus, Hinxton,

STN INTERNATIONAL LOGOFF AT 17:35:50 ON 03 NOV 2004